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PROCEEDINGS
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*ANTIBIOTIC SUBSTANCES FROM BASIDIOMYCETES IX. DROSOPHILA SUBATRATA. (BATSCH EX FR.) QUEL.**

By Frederick Kavanagh,† Annette Hervey and William J. Robbins

Department of Botany, Columbia University and The New York Botanical Garden

Communicated April 4, 1952

Drosophila subatrata[1] was found earlier in this laboratory to evidence substantial antibacterial activity when grown on agar media or in corn-steep liquid media.[2] In further investigation culture liquids of the fungus grown in Fernbach flasks in a corn-steep medium[3] at 25°C. developed an activity of 1000 dilution units or more against *Staphylococcus aureus*. After reflooding the fungus mat with fresh corn-steep medium high activity was obtained in 6 to 15 days.

Concentrates prepared from the culture liquid by extraction with methylisobutyl ketone or chloroform had activities against *Staph. aureus* of from 1000 to 6400 dilution units per mg. However, the stability of antibacterial activity of culture liquid and concentrates was variable. The data obtained on loss of activity when the concentrates were incubated with blood and on toxicity to mice by intravenous injection were inconsistent. Further investigation showed the presence in the culture liquid of at least four antibacterial substances which differed in stability, activity and other properties.

Isolation of Active Substances.—An activity of 1000 dilution units per ml. for *Staph. aureus* was obtained in the corn-steep medium in two weeks in shake culture. However, liquid from still cultures in the corn-steep medium was used for the isolation of the active substances as follows:

The culture liquid was acidified to between pH 3 and 4 and extracted with about one-tenth volume of chloroform. The chloroform was extracted with about one-tenth volume of 0.5 *M* sodium bicarbonate solution (*first* bicarbonate extract). About 37 per cent of the solids and 20 per cent of the activity against *Staph. aureus* were removed by the bicarbonate.

Two crystalline antibacterial substances (drosophilin A and B) were prepared from the chloroform after extraction with the bicarbonate by the

following procedure. The extracted chloroform was evaporated to about one-tenth volume in a flash-evaporator. The resulting dark brown concentrate was extracted with about an equal volume of 0.5 *M* sodium bicarbonate (*second* bicarbonate extract). This bicarbonate extraction removed about one-fourth of the antibacterial activity in the chloroform concentrate as measured against *Staph. aureus* and about 3 per cent of the solids. The second bicarbonate solution contained a highly active substance (later called drosophilin C).

Drosophilin A.—The chloroform concentrate after extraction with bicarbonate was further extracted with 0.5 *M* pH 10 carbonate buffer which removed a small amount of an active substance (drosophilin A) with a characteristic absorption spectrum (peak at 301 mμ). Crystalline drosophilin A was isolated from the carbonate buffer by acidifying and extracting with ether; pale yellow crystals formed when the ether evaporated. About 100 mg. of these crystals were obtained from 31 liters of culture liquid.

The crystals were purified by solution in benzene and treatment with decolorizing carbon. The white crystals obtained were recrystallized from hexane or from dilute aqueous alcohol; they melted at 118°C. (corr.) and remelted at the same temperature. The specific rotation was less than 0.5. Drosophilin A was soluble in hexane, benzene, acetone, alcohol, chloroform, ether and slightly soluble in water. It was not extracted from ether solution by concentrated HCl which indicated lack of basic properties. It was weakly acidic since it was not removed in appreciable amounts by extraction of the chloroform solution with bicarbonate but was removed by extraction with carbonate pH 10 buffer.

Drosophilin A was analyzed for C, H, methoxyl and Cl, and the oxygen found by difference; the molecular weight was determined cryoscopically in exaltone.[4]

	C	H	Cl	O	METHOXYL	MOL. WT.
Found	32.54	1.71	52.68	13.07	11.70	249
Computed for $C_{13}H_9O_4Cl_7$	32.70	1.90	51.99	13.41	13.00	477
Computed for $C_7H_4O_2Cl_4$	32.10	1.54	54.15	12.22	11.85	262

The empirical formula $C_{13}H_9O_4Cl_7$ fits the analysis better than $C_7H_4O_2Cl_4$ but not the molecular weight. Further work on drosophilin A is being undertaken.

Drosophilin A gave the same color reaction with alcoholic ferric chloride as pentachlorophenol. The absorption peak (301 mμ) was in the region of absorption by chlorinated phenols. It did not precipitate with silver nitrate; it reduced alkaline permanganate; it reduced alkaline silver

nitrate with the formation of silver chloride. It did not react with 2,4-dinitro-phenyl hydrazine and did not give reactions for α,β unsaturated ketones. Infra-red analysis[5] indicated the presence of hydrogen attached to unsaturated carbon, a hydroxyl group and a methyl or methylene group.

Drosophilin B.—The chloroform concentrate after extraction with pH 10 buffer was extracted with 0.5 *M* sodium carbonate solution which removed a small residue of drosophilin A and colored substances. The chloroform was then extracted with 1 *N* KOH solution (which removed a small amount of colored substances), washed with water and acid and evaporated to dryness. The treatment with carbonate and hydroxide removed 17 per cent of the solids. The solids obtained by evaporation of the chloroform were dissolved in benzene and evaporated slowly. The crystals (drosophilin B) which formed were recrystallized from benzene and from dilute aqueous alcohol. The melting points, mixed melting points, analyses, molecular weight, infra-red and ultra-violet spectrum, solubilities, optical activity and the general chemical and biological properties showed drosophilin B to be identical with pleuromutilin.[6] The infra-red spectrum indicated the presence of hydroxyl, carbonyl and possibly an ester group.

TABLE 1

POSITION OF ABSORPTION PEAKS IN mμ AND OPTICAL DENSITY FOR ALCOHOLIC SOLUTIONS OF DROSOPHILINS C AND D

C	207(0.72)	234(0.41)	269(0.31)	281(0.33)	
D	217(1.42)	259(0.33)	274(0.54)	290.5(0.76)	309(0.61)

Drosophilin C.—This substance which had the greatest antibacterial activity was obtained from the second bicarbonate extract. Since it was inactivated by drying in air it was necessary to avoid drying during isolation.

The acids in the bicarbonate extract were transferred to ether and the ether solution was shaken repeatedly with one-fourth volume of 0.1 *N* silver nitrate.[7] The yellow precipitate was removed each time and kept under water. The combined silver precipitates were washed with ether and water, ether added, the silver compound decomposed by addition of HCl and the organic acids transferred to ether. The substances in the ether were further purified by counter-current distribution between chloroform and 0.5 *M* phosphate buffer of pH 6.9. Most of the antibacterial activity and the solids were in the chloroform which had traveled through the system. This chloroform solution was distributed again in a four-funnel system using buffer of pH 7.5. The purest substance was found in the third funnel. A total of 4 mg. of drosophilin C was obtained from 31 liters of culture liquid. Probably at least as much was in the other fractions from the counter-current distribution system; the chemical properties indicate that drosophilin C should be found in other fractions and the

treatment with silver nitrate did not remove all of it from the ether solution.

The absorption spectrum was measured in alcoholic solution and the results reported (table 1) as optical density (in parentheses) for solutions with a concentration of 10 μg. per ml. in 1-cm. thick cuvettes.

Drosophilin D was obtained from the first bicarbonate extract. The extract was acidified and the acids extracted into chloroform. The chloro-

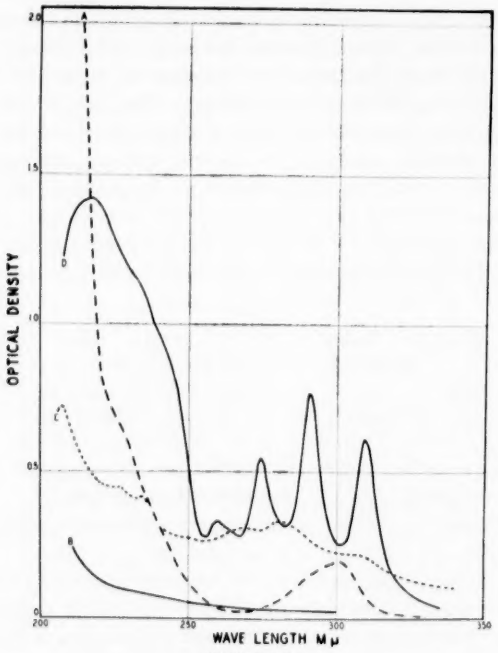

FIGURE 1

Absorption spectra of drosophilins A, C, and D measured in ethanol at a concentration of 10 μg. per ml. and B at a concentration of 100 μg. per ml.

form was shaken with one-fourth volume of pH 6 buffer which removed most of the colored substances but only traces of antibacterial activity. The chloroform was then extracted repeatedly with silver nitrate with removal of the precipitated silver compound before repeating the treatment with fresh silver nitrate. About three-fourths of the activity of the bicarbonate solution was lost at this step and most of that remaining was in the chloroform. The chloroform solution was treated to a counter-current

distribution using pH 7.2 buffer. A substance with an ultra-violet absorption spectrum[8] characteristic of polyacetylene substances was found in largest amount in the middle funnels. The distribution was repeated and material from the middle funnel of a five-funnel system was taken as representative of the active substance. This compound, drosophilin D, was soluble in organic solvents, somewhat soluble in water, was precipitated from aqueous alcoholic solution by silver nitrate as was drosophilin C. Drosophilin D titrated in 20 per cent ethanol as a monobasic acid with a pK of 4.2 and an equivalent weight of 405.

The absorption spectrum (table 1 and Fig. 1) was determined in the same way as for drosophilin C. The frequency differences of the position of the absorption peaks of drosophilin D are 224.2, 63.4, 62.2 and 61.8*f*. The latter three differences are characteristic of polyacetylenes.[8] Drosophilin D is the sixth polyacetylene (identified in this laboratory) which has antibacterial activity and is produced by species of Basidiomycetes.[9]

TABLE 2

MINIMUM INHIBITORY CONCENTRATION IN μg. PER ML.

BACTERIA	DROSOPHILIN A	B	C	D
Bacillus mycoides	64	125	16	125
Bacillus subtilis	32	16	0.12	2
Escherichia coli	250	250	16	250
Klebsiella pneumoniae	64	0.5	32	500
Mycobacterium smegmatis (smegma)	64	8	16	64
Pseudomonas aeruginosa	250	500	32	250
Staphylococcus aureus	4	0.5	0.03	4

Antibacterial Activity.—The activity of these four compounds against seven bacteria was measured by the methods used in this laboratory.[10] The substances were active mainly against Gram-positive bacteria.

Some culture filtrates had an antibacterial activity consistent with the assumption that most of the activity was due to drosophilin C.

The activity of the drosophilins was tested against 60 bacteriophages.[11] Drosophilin A was active against only staphylophage 8, drosophilin B (pleuromutilin) against streptophage 6 and 7; the other two drosophilins were inactive. Observations of the antibacterial activity were made incidental to the determination of antiphage activity. Drosophilin A was active against the strains of staphylococci and *B. subtilis* but inactive against streptococci and Gram-negative bacteria. Drosophilin B was active against most of the strains of bacteria. Drosophilin C showed some activity against all Gram-positive bacteria. Drosophilin D was active against all of the bacteria with activity greatest against Gram-positive bacteria.

Effect of Blood.—When the four drosophilins were incubated for four

hours with 5 per cent human blood in beef extract test medium, A and B were not inactivated, C was $^{7}/_{8}$ inactivated and D lost one-half of its activity. The inactivation of some culture filtrates by blood suggested that most of the activity was caused by drosophilin C.

Animal Toxicity and In Vivo Tests.[12]—Drosophilin B (pleuromutilin) was administered in single doses immediately after infecting mice with *Diplococcus pneumoniae* (ATCC 6302). The mice receiving the largest dose (1 mg.) did not live longer than the untreated controls. Drosophilin B had an *in vitro* activity of 3 μg. per ml. against *D. pneumoniae*. Drosophilin B was not toxic to the mice when given at 1 mg. to 20 g. mice by either the intravenous or the intraperitoneal routes. The lack of animal toxicity and the ineffectiveness of drosophilin B was another indication of its identity with pleuromutilin.

* This investigation was supported in part by the generosity of William Bingham II and by a grant from the National Microbiological Institute, Federal Security Agency, Public Health Service.

† Present address, Commercial Solvents Corporation, Terre Haute, Ind.

[1] The culture of the organism was obtained from Dr. José Emilio Santos Pinto Lopes, Instituto Botanico, Lisbon, Portugal, to whom the authors express their thanks. The fungus is also known as *Agaricus subatratus* Batsch ex Fr. and *Psathyrella subatrata* (Batsch ex Fr.) Gell.

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[3] Robbins, W. J., Kavanagh, F., and Hervey, Annette, These PROCEEDINGS, **33,** 171–176 (1947).

[4] The quantitative analyses and molecular weight determinations were made by the Huffman Microanalytical Laboratories, Denver, Colo.

[5] Infra-red spectra and interpretations were made by Dr. H. L. Dinsmore, Commercial Solvents Corporation.

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[7] Purification through the silver salt was also used in purifying biformin which had chemical properties similar to drosophilin C, These PROCEEDINGS **33,** 176–182 (1947).

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[12] The authors are indebted to H. Payne and R. Baldwin, Commercial Solvents Corporation for these determinations.

ARGINOSUCCINIC ACID FROM CHLORELLA

BY JAMES B. WALKER

FROM THE BIOCHEMICAL INSTITUTE AND THE CHEMISTRY DEPARTMENT OF THE UNIVERSITY OF TEXAS, AND THE CLAYTON FOUNDATION FOR RESEARCH, AUSTIN, TEXAS

Communicated by Roger J. Williams, May 1, 1952

Artificially induced enzyme blocks are invaluable in elucidating metabolic pathways. Lyophilization or desiccation with acetone was found to inactivate some of the enzyme systems of *Chlorella pyrenoidosa* cells; consequently it was hoped that a systematic study of the inhibited reactions and the alterations of metabolism resulting from such treatment might furnish a better understanding of the intermediary metabolism of algae. Out of this study has developed the isolation and characterization of arginosuccinic acid, a compound of unusual properties and significance for intermediary metabolism.

Experimental.—*Chlorella* cells were grown photoheterotrophically at room temperature in 1-liter cotton-stoppered Erlenmeyer flasks on a mechanical shaker, with 4% glucose as the carbon source and 0.4% urea as the nitrogen source. Cells were harvested by centrifugation, washed twice, acetone-dried or lyophilized, and stored in the refrigerator; 6 to 8 g. of dried cells were obtained per flask.

When a 5% aqueous suspension of acetone-dried cells was shaken at room temperature on a mechanical shaker with fumarate or malate (final concentration 0.1 *M*), an amino acid was produced and excreted into the medium in quantities readily detectable within twenty minutes, although production continued for several hours. This compound could be distinguished from all common amino acids by two-dimensional paper chromatograms, employing as solvents water-saturated phenol, ammonia atmosphere (R_f 0.27), followed by a 4:1:1 *n*-butanol-acetic acid-water mixture (R_f 0.08). A purple spot was obtained when the papergrams were sprayed with ninhydrin, while a more specific but less sensitive color reagent, alkaline picrate, produced an orange spot.

Similarly treated cells from cultures grown with nitrate instead of urea as the sole nitrogen source did not produce this substance unless arginine, in addition to fumarate or malate, was added to the suspension of acetone-dried cells. Acetone-dried cells prepared from cultures grown in a urea medium have a much higher arginine content and did not require exogenous arginine to produce the new compound (arginosuccinic acid), but arginine addition did increase the rate of formation, as well as the final yield. Arginine, when added singly to a suspension of acetone-dried cells grown on either urea or nitrate, was transformed into citrulline, as in the work with bacteria recently reported by Schmidt, *et al.*[1] and Knivett,[2]

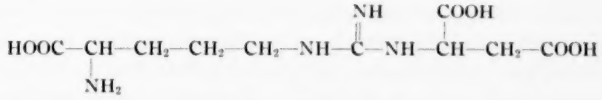
but when sufficient malate or fumarate was also added, arginosuccinic acid was formed instead of citrulline.

In arginosuccinate production arginine could not be replaced by citrulline, ornithine, creatine, or guanidine, while the role of fumarate-malate could not be played by succinate, aspartate, glutamate, citrate, lactate, pyruvate, acetate, butyrate, glycolate, phosphoglycerate, tartrate, maleate, ethanol, etc.; oxalacetate had a small but definite amount of activity. Preliminary time course experiments with cyclohexanol as a partial fumarase inhibitor indicated that fumarate rather than malate may be the immediate precursor, because with equal substrate concentrations the arginosuccinate ninhydrin spot was less intense with malate than with fumarate. When one reviews known reactions in which fumarate will act but malate will not, the addition of ammonia to fumarate to form aspartic acid comes to mind; in an analogous manner arginine might add to fumarate to form arginosuccinic acid.

To produce sufficient quantities of arginosuccinate so that it could be isolated, acetone-dried cells (cultured in urea) were shaken under toluene with arginine and malate for 10 hours, after which time the cells were centrifuged off and recharged with fresh substrates; recharging at intervals was continued until 40 hours had elapsed, after which time the cells were discarded. The combined supernatants were concentrated, deproteinized, and passed through a Dowex-50 column with 4 *N* HCl as eluant. Removal of the HCl *in vacuo* from the fractions containing arginosuccinic acid left a mixture of the latter, large amounts of KCl, and some arginine. An alcohol extract of this residue was next passed through another Dowex-50 column and eluted with 2.5 *N* HCl. Removal of the HCl as before gave a very hygroscopic white solid, which papergrams showed contained only an estimated 5% ninhydrin reactive impurity (arginine). The solid was dissolved in alcohol and filtered; slow addition of a dilute alcoholic solution of aniline to the filtrate gave a white precipitate, which was washed several times with alcohol and immediately dried in a vacuum desiccator. This precipitate was relatively non-hygroscopic and free from other ninhydrin reactive compounds; its temperature of decomposition was approximately 270°C. Although insoluble in alcohol, it dissolved readily on addition of HCl; from aqueous solution it gave a white precipitate with silver nitrate in the presence of nitric acid.

Three-day hydrolysis of arginosuccinic acid with 6 *N* HCl at 100°C. gave arginine as the only ninhydrin reactive product, while a 12-hour 4 *N* barium hydroxide hydrolysate showed two ninhydrin spots corresponding to ornithine and aspartate.

These data are best explained by assuming that this compound is that postulated and partially characterized by Ratner[3] as the intermediate in the synthesis of arginine by mammalian liver from citrulline plus aspartate:



It is likely that arginosuccinic acid is identical also with the unknown compound of Davison and Elliott[4] produced on incubation of various tissues with arginine plus fumarate, since the precursors are identical and the R_f values appear similar.

Microbiological assays were next employed in order to obtain quantitative data which might throw further light on the structure and purity of the isolated arginosuccinic acid. The organisms used were *Leuconostoc mesenteroides* P-60 for arginine and aspartate assays, and *Escherichia coli* B, mutant No. 43, for ornithine and arginine assays. The latter organism[5] was found to require for growth ornithine, citrulline, or arginine, on an equimolar basis, in addition to the glucose-asparagine-salts minimal medium.

Unfortunately, neither of these two organisms nor *Lactobacillus casei* was able to use the intact arginosuccinic acid molecule to supply its aspartate or arginine requirement; thus direct microbiological assay with these organisms was impossible.

Since these organisms fail to utilize arginosuccinate unless it is "predigested" for them, it was necessary to employ chemical hydrolysis and analyze for the fragments. Ten milligrams of the hygroscopic column isolate hydrolyzed three days with 6 *N* HCl gave, by both *E. coli* and *L. mesenteroides* assay, the same amount of L-arginine, 2.1 mg. (calculated as the monohydrochloride). When an aliquot of this acid hydrolysate was further subjected to barium hydroxide hydrolysis no aspartate was formed, but DL-ornithine calculated equivalent to 4.1 mg. of DL-arginine monohydrochloride from the original sample was produced. This when taken in conjunction with the 2.1 mg. of L-arginine monohydrochloride found above indicates that the arginine moiety in arginosuccinic acid had been racemized. The alternative possibility that both arginine and guanidosuccinic acid were formed by acid hydrolysis was ruled out by the lack of aspartate production upon subsequent alkaline hydrolysis. Since alkaline hydrolysis is faster and produces two analyzable fragments instead of one, it was the analytical method of choice for studies of per cent purity.

A sample of purified, vacuum-dried arginosuccinic acid, weighing 18.9 mg. and found by paper chromatography to be free from other amino acids, was hydrolyzed for 20 hours with 4 *N* barium hydroxide. The hydrolysate was found to contain by *E. coli* assay the equivalent of 10.0 mg. of DL-ornithine monohydrochloride and by *L. mesenteroides* assay 5.1 mg. of DL-aspartic acid. Assuming the purified arginosuccinic acid to be in the monohydrochloride form, this assay showed that from each mole of arginosuccinic acid were produced 1.02 moles of ornithine and 0.66 mole of

aspartic acid. These results can be explained by assuming that 0.34 mole of arginosuccinic acid underwent hydrolytic cleavage to form arginine before subsequent conversion to ornithine, while 0.66 mole was cleaved into aspartic acid, ornithine, carbon dioxide and ammonia, with perhaps citrulline and ureidosuccinate as transitory intermediates in the process. The per cent purity calculated from the moles of ornithine liberated gives a value of 102%.

Discussion.—Ratner[3] obtained the barium salt of the intermediate formed by liver from citrulline and aspartate, and she estimated that her sample was 81% pure from its barium content, assuming 1.5 moles of barium per mole of intermediate.

Larger amounts of arginosuccinic acid can be produced with less trouble from acetone-dried *Chlorella* (or *Scenedesmus*), however, and no complicated enzyme isolation is required. The procedure described here leading to the production of the pure compound, together with the chromatographic, microbiological, and colorimetric characterization techniques, should prove helpful for further investigations of the role of arginosuccinic acid in metabolism.

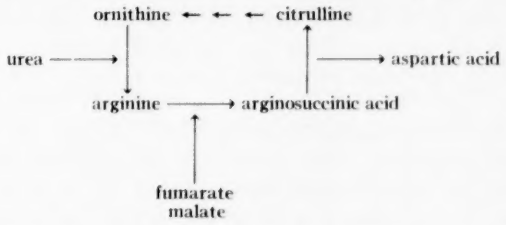
Several puzzling aspects of this problem remain to be worked out, however. Knivett[2] reported that the formation of citrulline from arginine was inhibited by disubstituted guanidines, among other compounds; this would perhaps explain why arginine added singly to acetone-dried cells yields citrulline, while the addition of fumarate or malate along with the arginine inhibits the formation of citrulline. The disubstituted guanidine, arginosuccinate, which is formed might inhibit in higher concentrations an enzyme involved in one of the subsequent transformations usually occurring during arginine metabolism; this could be yet another example of the intricate mechanisms by means of which alternate metabolic pathways are selected as the physiological environment varies.

The failure of the three organisms tested to grow on the intact arginosuccinate molecule does not preclude the possibility that a more determined search will turn up organisms with which a direct assay can be made. Since toxicity was ruled out by suitable controls, the lack of biological activity of the intact molecule might be ascribed to configurational properties, permeability restrictions, or to inter- or intramolecular cyclization. That at least some of the molecules may be cyclized is perhaps indicated by the fact that the color with alkaline picrate is similar to that with creatinine, both on papergrams and in solution; however, color reactions are often unpredictable, and it may be that the disubstituted guanidine structure of arginosuccinate as written could produce such a color with alkaline picrate. The time characteristics of the intensity of the color developed with alkaline picrate are quite critical; the optimum conditions for quantitative determinations of arginosuccinic acid by this method are currently being worked out.

The uniqueness of arginosuccinate in intermediary metabolism is readily apparent when one examines the many possible products of known biochemical importance which might arise from degradation of this compound. Such products include arginine, citrulline, ornithine, fumarate, malate, ureidosuccinate and aspartate. By controlling the available metabolic pathways leading to and from this compound alone, the organism might display a variety of responses.

From contemplation of its structure, arginosuccinic acid appears to be a logical pyrimidine precursor; the work of Wright, *et al.*,[6] establishing ureidosuccinate as a pyrimidine precursor furnishes experimental support for such speculation. Organisms requiring both arginine and a pyrimidine for growth might be used to test this hypothesis. Should arginosuccinate indeed be found to be a pyrimidine precursor, investigations directed toward finding specific inhibitors of that metabolic pathway might prove fruitful as a means of controlling cell division. Inhibition of pyrimidine synthesis near the arginosuccinate level should be less likely to lead to the accumulation of toxic products, since this compound could be readily diverted into other pathways.

Another possible function of arginosuccinate with widespread implications is as an intermediate in urea nitrogen assimulation in cells lacking urease. No measurable urease activity could be found in *Chlorella*, and measurements and calculations showed that non-enzymatic hydrolysis under the experimental conditions employed was not appreciable. In view of the large production of arginine by cells grown with urea as the nitrogen source, it is attractive to suppose that urea might enter the metabolic pool via a reversal of the mammalian-mold urea cycle.



If such a mechanism exists, the initial coupling of urea with ornithine, by analogy with that of citrulline with aspartate,[3] would presumably require ATP.

Acknowledgment.—This investigation was made possible by the generosity of Professor Jack Myers, in whose Laboratory of Algal Physiology this work was performed.

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*THE MECHANISM OF POLYMERIZATION OF FIBRINOGEN**

By John D. Ferry

Department of Chemistry, University of Wisconsin

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Introduction.—Fibrinogen, a rod-like protein of length about 600 Å and molecular weight about 350,000, polymerizes under the catalytic action of thrombin to form fibrin, a three-dimensional network structure. A network strand, as seen under the electron microscope, often exhibits uniform width and little curvature over lengths many hundred times that of fibrinogen; the strand width may range from double to many times that of fibrinogen (about 30 Å), depending on the pH and ionic strength and other conditions. There is good evidence that the fibrinogen units retain their identity within the strands and that their long axes are aligned in the strand direction.

Two intermediates in the polymerization process have been recently identified. The first (**f**) accumulates when thrombin activates fibrinogen at abnormally low pH (about 5),[1] or at abnormally high ionic strength; it has the same gross size and shape as fibrinogen itself, though it has probably suffered the loss of one or more small fragments in the course of activation;[2] it polymerizes rapidly when returned to the normal pH and ionic strength. A similar molecule is formed by dissolving fibrin in 3.5 *M* urea[3] or 2 *M* lithium or sodium bromide.[4] The second intermediate ($\mathbf{f}_n$) accumulates when thrombin activates fibrinogen in the presence of an inhibitor such as 0.5 *M* hexamethylene glycol,[5] 0.5 *M* lithium bromide, or 1 *M* urea.[6] It appears to be a partial polymer of **f** and is so symbolized; **f** can be converted to $\mathbf{f}_n$ and vice versa by changing the solvent composition;[6] and $\mathbf{f}_n$ dissociates to form **f** simply upon dilution.[5,7] The conversion of fibrinogen (*F*) to fibrin is thus pictured as follows:

$$F \xrightarrow{T} \mathbf{f} \rightleftarrows \mathbf{f}_n \rightarrow \text{Fibrin}$$

The intermediate polymer $\mathbf{f}_n$ may be regarded as a sort of prototype of a fibrin strand. Its size and shape have been studied by viscosity and sedimentation,[5,6] flow birefringence,[8,9] and light scattering.[7] In 0.5 *M*

hexamethylene glycol, ionic strength 0.45, and pH 6.2, at 25°C., its cross-section area is about double and its length ranges from 6 to 10 times that of fibrinogen, with a weight-average degree of polymerization of about 15. These figures are consistent with electron microscope observations of preparations from inhibited clotting systems. Such a polymer could be formed by a lateral dimerization process with partial overlapping, resulting in two parallel and end-to-end chains with staggered junctions.

Significance of the Intermediate Polymer.—It is easy to see how an inhibitor which blocks sites of attractive force could limit the polymerization,[10] but it has been difficult to understand why the growth should be stabilized at an intermediate stage with the comparatively narrow distribution of values of n which the flow birefringence and sedimentation data indicate. This and other relations between fibrinogen and fibrin might be explained by a more detailed consideration of the attractive and repulsive forces between molecules of the monomer intermediate, **f**.

The reaction of F with thrombin has evidently exposed sites of strong attractive forces, which may be partly dipolar (probably of particular significance in orienting the rods suitably for interaction) and partly due to hydrogen bonds and the association of non-polar groups. However, within the pH range where clotting occurs, **f** carries a net negative charge.[11,12] As successive monomer molecules are added to a growing polymer, increasing electrostatic work must be done. Without inhibitors, this is readily surmounted by the free energy loss due to local attractive forces when two **f** molecules come into contact; at neutral pH and normal ionic strength the degree of polymerization is therefore unlimited, though above pH 9 the rate of clotting is reduced by electrostatic repulsion and above pH 10 the net charge is high enough to prevent clotting from going to completion.[11]

Hexamethylene glycol and other inhibitors evidently reduce the local attractive forces, however, so that the local (negative) free energy and long-range electrostatic (positive) free energy of association are of the same order of magnitude, and their sum goes through a minimum with increasing n; the situation is analogous to the formation of a soap micelle as treated by Debye,[13] except for difference in geometry. This interpretation explains why the arrested polymer has a degree of polymerization rather narrowly distributed around a characteristic value of n, but still not exactly monodisperse. It also predicts the presence of a "critical micelle concentration" below which $\mathbf{f}_n$ is unstable and dissociates to **f**; this has been in fact observed by light-scattering measurements.[7] Under the experimental conditions mentioned above, where $n = 15$, the critical concentration is about 0.5 g./l. in terms of the total protein participating in the equilibrium. The fraction of protein in the polymeric form, plotted against concentration, rises sharply at this point, just as in the formation

of soap micelles.[14] The critical concentration corresponds to a standard free energy of formation of $\mathbf{f}_n$ of about $-120{,}000$ calories per mole, or -8000 calories per monomer unit.

Relation of Attractive Forces to Specific Groups.—To explain why **f** does not polymerize at all at pH 5—even though the net charge here is slight—it must be assumed that the local attractive forces disappear. The marked difference between pH 5 and pH 6 (which is also reflected in the clotting time,[11] a measure of the over-all rate of conversion of fibrinogen to fibrin) can be attributed to the ionization of a specific group on the molecule. There are only two groups commonly present in proteins which dissociate in the appropriate range. One is histidine, previously proposed in connection with the pH dependence of the clotting time.[11] The other is a phosphoric ester, the presence of which in fibrinogen, however, has not been reported. It is suggested that the formation of a bond between two **f** molecules involves either a neutral histidine residue or, less likely, a phosphate group with two negative charges. This hypothesis bears also on the dissolution of fibrin in concentrated solutions of lithium salts,[4] guanidinium chloride,[6] histamine dihydrochloride[6] and urea.[6,12] While urea and guanidine have a general dispersive action on proteins and at extremely high concentrations denature, they may also alter the ionization of dissociable groups through specific interaction.

Another specific group has been identified by Bailey and Lorand,[15] who have shown that several terminal amino groups of glycine are liberated by thrombin. Waugh[16] has concluded from an analysis of kinetics of clotting that each fibrinogen molecule experiences only one encounter with thrombin. Our picture of lateral dimerization implies that the loci of attraction are on the sides of the molecules. We may postulate that thrombin reacts near the center of one side and leaves a concentration of positive charge in the form of terminal amino groups. Then if there is a concentration of negative charges near the ends of the fibrinogen molecules the conditions are fulfilled for orientation, by electrostatic forces, in exactly the overlapping dimerization configuration which has been deduced from the dimensions of the intermediate polymer. If the critical histidine groups are near the ends, their acquisition of positive charges at low pH would destroy the electrostatic pattern.

Although under normal physiological conditions the growth of protofibrils $\mathbf{f}_n$ should be practically unlimited, the local geometry of polymerization is very probably the same as that prevailing in the inhibited systems which can be studied experimentally. The final step in the formation of fibrin is then the association of protofibrils in bundles to form the strands seen in the electron microscope. It is presumably at this stage that the differences between "coarse" and "fine" clots[17] are introduced, the width of the bundles depending on the pH and ionic strength.

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*PSEUDO-WILD TYPES IN NEUROSPORA CRASSA**

BY M. B. MITCHELL, T. H. PITTENGER AND H. K. MITCHELL

KERCKHOFF LABORATORIES OF BIOLOGY, CALIFORNIA INSTITUTE OF TECHNOLOGY, PASADENA, CALIFORNIA

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In Neurospora the test for non-allelism which is usually considered conclusive consists of the recovery of the double mutant from a cross between the two mutants in question and the demonstration that the two parent mutants segregate in asci from a cross of the double mutant to wild type.[1] Carrying out this test is usually a simple matter if fertile crosses can be obtained and if close linkage between the two mutants does not exist. Crosses between two pyrimidine mutants to be considered here, however, gave no complete asci and only a small percentage of phenotypically wild progeny. Since the mutants are not distinguishable by known physiological tests, the double mutants, if they occurred, could presumably be detected only by out-crossing mutant progeny at random and examining asci. This procedure seemed impracticable, and, instead, phenotypically wild progeny were studied with the possibility in mind that they might be found

to differ from standard wilds in a way which would indicate that they did not arise from crossing-over between the mutant loci. Such a difference was readily demonstrated by crossing these strains to standard wild, since these crosses regularly gave about 45% pyrimidine mutant progeny. Strains which behaved in this fashion have been termed pseudo-wilds, since they appear to be wild phenotypically, but give mutant progeny. Pseudo-wilds from several other crosses have been isolated and studied in an attempt to explain the origin and behavior of these strains.

Mutant Strains from Which Pseudo-wilds Were Derived.—The three pyrimidine mutants which were used in these experiments are *pyr-3a* (37301),[2, 3] *pyr-3b* (37815)[2] and *pyr-3d* (45502).[4, 5] Mutants *pyr-3a* and *-3d* require pyrimidine at any temperature, but *pyr-3b* has an absolute requirement only at temperatures above about 32°C., being phenotypically wild at 25°C. There is no evidence at present that these three mutants are non-allelic. The formation of pyrimidine-independent heterocaryons in mixed cultures of *pyr-3a* and *-3d* has not been observed although several isolates of each mutant have been tested. Pseudo-wilds have been found from *3a* × *3d* and *3b* × *3d* but not, so far, from *3a* × *3b*. Two other mutants, closely linked to these, have been used, the "colonial," *co* (70007),[6] and the arginine mutant, *arg* (33442).[7] The isolate of *pyr-3d* first studied carried a translocation[4, 8] to which the mutant gene is linked. Isolates of this mutant with and without the translocation have been used. When the translocation is absent, crosses to wild and to certain other mutants give predominantly normal asci, but when it is present many asci are found with defective spores. Some contain only defective spores and others contain both normal and defective spores in pairs. The mutant without the translocation was found in an ascus from a cross of *pyr-3d* T to *co*. The strain carries mutant *co* and is designated *co pyr-3d*. The absence of the translocation has not been demonstrated cytologically but is deduced from the observations that asci showing defective spores in pairs are rare from crosses involving this strain but were found very frequently from those involving strains from two other spore pairs from the same ascus. Also, the centromere distance of *pyr-3d* calculated from 19 asci from *co pyr-3d* × *co* is 13 units, whereas that calculated from 104 asci[5] from crosses involving *pyr-3d* T is one unit. A value for *pyr-3a* based on 401 asci[5] (an unpublished data) is 14 units.

With a few exceptions progeny of crosses reported here were analysed by classifying and counting random spores plated on minimal medium, or minimal supplemented with cytidine or arginine. Spores were collected from the wall of the test tube in which the cross was made, suspended in 1 ml. of minimal medium which was then spread on the agar surface. The spores were heat-treated on the plates and incubated at 25°C., except those from crosses involving *pyr-3b* which were incubated at 34°C. After 12 to

20 hours the germinated spores were classified under the microscope. The *arg* and *pyr* spores can be distinguished from wild type because of the limited growth of their hyphae on minimal medium. "Colonial" can be classified by its growth pattern which is quite different from that of wild type. It can be classified on supplemented medium when it is combined with *arg* or *pyr*. From crosses involving the five mutants, counts of spores plated in minimal medium are given below.

	CROSS	WILD AND/OR PSEUDO-WILD	*pyr* AND/OR *arg*	*co*
1	*co* × *pyr-3a*	21 = 1.2%	891	805
2	*co* × *pyr-3d* T	265 = 4.7%	2,613	2693
3	*co* × *arg*	13 = 0.4%	1,648	1592
4	*arg* × *pyr-3b*	12 = 0.6%	2,125	..
5	*pyr-3a* × *pyr-3b*	0	20,888	..
6	*pyr-3a* × *pyr-3d* T	60 = 4.6%	1,259	..
7	*pyr-3b* × *pyr-3d* T	57 = 3.8%	1,448	..
8	*pyr-3b* × *pyr-3b*	0	16,607	..
9	*pyr-3a* × *co pyr-3d*	81 = 1.0%	8,034	0
10	*pyr-3b* × *co pyr-3d*	4 = 0.14%	2,913	0
11	*arg* × *co pyr-3d*	7 = 0.15%	4,549	49

Crosses 1, 3, 4, 5, 8 and 11 gave many asci with eight normal-appearing spores but germination was not always good. Cross 2, as already mentioned, gave many asci with aborted spores (pale, or small and colorless). Asci with eight normal-appearing spores have not been observed from crosses 9 and 10. Most of the spores appear to be of normal size but many do not darken normally. The degree of darkening, even among members of pairs, is variable and those which are not fully darkened rarely germinate. Spores from crosses 6 and 7 exhibit this type of abnormality as well as the spore abortion accompanying the translocation. Of the *pyr* mutants only *pyr-3b* could be crossed to itself successfully. Mutants *pyr-3a* and *pyr-3d*, when selfed, formed perithecia but normal spores were not observed. Spores from the crosses *pyr-3a* × *pyr-3b* and *pyr-3b* × *pyr-3b* were incubated at 34°C. for 2 or 3 days before they were counted.

Pseudo-wilds from pyr-3a × *pyr-3d T.*—From two crosses involving different isolates of *pyr-3a* and *-3d* T, 40 phenotypically wild spores were transferred from minimal plates to slants of minimal medium. The resulting strains were subcultured on minimal medium and on minimal with cytidine. Ten strains from one cross were tested in 125-ml. flasks containing 20 ml. of liquid minimal medium. Each strain was tested after being subcultured twice on minimal and twice on minimal with cytidine. Mycelial pads were weighed after a growth period of 4 days at 25°C. The lowest, highest and average dry weights, in mg., for each set of 10 cultures are given below

	LOWEST	HIGHEST	AVERAGE
Subcultured on minimal	39	74	62
Subcultured on cytidine	34	80	66

A second group of 10 strains from the other cross was tested after being subcultured four times on minimal. Dry weights after a 3-day growth period were as follows: lowest—21; highest—49; average—41. At the same time corresponding dry weight determinations were made from flask cultures in which the minimal medium was supplemented with 1 mg. of cytidine sulfate per flask. These weights did not differ significantly from those given above. Thus, in their growth on minimal medium and in their failure to be stimulated by cytidine, these strains resemble closely wild types isolated at random. In other experiments, however, 12 strains derived from phenotypically wild, germinated spores which were transferred to Westergaard's basic medium[9] supplemented with an autolysate of wild-type Neurospora mycelium, and subcultured on this medium, showed stimulation by cytidine in the early stages of growth in liquid culture. This behavior suggested that the strains were not genetically pure and that a pyrimidine-dependent component was selected.

Crosses of the 40 strains mentioned above (kept on minimal medium) to standard wild types, 7A and 8a, were attempted. In every case perithecia were formed with one or the other mating type, but in 25 cases no mature spores, or very few, were observed. Spores from the 15 more fertile crosses (7 of the strains involved were among those tested in flasks) were plated and counted with the following results:

WILD × PSEUDO-WILD	WILD	MUTANT	% MUTANT
7A × 6 R 22	988	804	45
8a × 6 R 31	740	589	44
7A × 6 R 32	759	625	45
8a × 6 R 36	916	660	41
7A × 6 R 38	666	489	42
7A × 6 R 41	715	578	45
8a × 6 R 42	711	556	44
8a × 6 R 43	713	542	43
7A × 6 R 45	468	444	49
7A × 6 R 46	236	156	40
7A × 6 R 48	460	428	48
8a × 6 R 50	796	691	46
8a × 6 R 55	887	695	44
7A × 6 R 56	422	323	43
8a × 6 R 57	908	747	45
	10,385	8327	44

From each cross 10 mutant-appearing spores were isolated and cultured on minimal with cytidine. Five did not grow further but the remaining

145, when tested in liquid culture, grew when supplied cytidine and failed to grow when not so supplied. Spores from a small number of asci (26 in all) from three crosses were isolated and tested. From each ascus four spores gave rise to *pyr* strains and four to wild type.

Pyrimidine mutant spores were recovered from crosses to wild of 5 of the 12 strains kept on the autolysate medium. Randum spores from these 5 crosses were plated but not counted.

Pseudo-wilds from pyr-3b × pyr-3d T.—Since *pyr-3a* and *-3d* are not distinguishable from each other, it was not possible to discover whether both parent mutants were recovered from out-crosses of pseudo-wilds. In order to answer this question, attempts were made to obtain pseudo-wilds from crosses involving *pyr-3b*. As shown above, phenotypically wild progeny have not been observed from *pyr-3a* × *pyr-3b*, but have been obtained from *pyr-3b* × *pyr-3d* T. Six strains from the latter cross were isolated and cultured on Westergaard's minimal. (There was no reason for using this medium instead of Fries, except that it was convenient.) On minimal medium dry weights, in mg., obtained from flask cultures kept at 34°C. for 3 days were as follows: 7 R 1—63; 7 R 3—68; 7 R 4—60; 7 R 5—49; 7 R 7—48; 7 R 9—75. When crossed to wild types 1400-4A and 1347-2a, each strain formed perithecia with only one mating type but three produced few spores. Spores from the three fertile crosses were plated and incubated at 25° and at 34°C. Counts appear below:

	WILD	MUTANT	% MUTANT
Wild a × 7 R 1 A 25°C.	857	799	48
Wild a × 7 R 1 A 34°C.	865	873	50
Wild a × 7 R 4 A 25°C.	550	549	50
Wild a × 7 R 4 A 34°C.	910	959	51
Wild a × 7 R 9 A 25°C.	1500 (approx.)	0	..
Wild a × 7 R 9 A 34°C.	1212	1241	50

Ten mutant spores isolated from the 7 R 1 cross proved to be phenotypically like *pyr-3d* and 14 from the 7 R 9 cross, plated at 34°C., were like *pyr-3b*. From this it appears that either one or the other of the parent mutants was recovered from these pseudo-wilds. Examination of asci from the three crosses showed the presence of defective spores in pairs in the 7 R 1 and 7 R 4 asci but not in asci from 7 R 9.

Pseudo-wilds from co pyr-3d Crosses.—It seemed possible that the pseudo-wilds might arise from an effect of the translocation. Therefore, when the *co pyr-3d* strain was obtained, phenotypically wild strains from *pyr-3a* × *co pyr-3d* were examined. When six such strains were isolated and cultured on Westergaard's medium it was observed that, although they had not been phenotypically *co* on the agar plate, as they grew they began to show this character in what may be called a diluted condition. The extent of

expression of *co* varied in different isolates and in different subcultures of the same isolate. The six strains gave the following dry weights, in mg., from flask cultures kept at 25°C. for 4 days: 9 R 2—48; 9 R 4—58; 9 R 6—55; 9 R 7—55; 9 R 8—54; 9 R 9—39. Crosses of the six isolates to wild were all fertile; spores were plated on minimal agar and classified as follows:

	WILD	*pyr*	*co*	% *pyr*
Wild A × 9 R 2 a	742	629	14	46
Wild A × 9 R 4 a	738	517	4	41
Wild a × 9 R 6 A	1006	789	12	44
Wild A × 9 R 7 a	788	557	4	41
Wild a × 9 R 8 A	717	532	2	42
Wild A × 9 R 9a	719	624	3	46

Germination of spores on these plates was not especially good, which may account for low percentages of *pyr* mutants.

The origin of pseudo-wilds, then, does not depend upon the presence of the translocation, but the behavior of these strains with respect to *co* suggests that they differ from those isolated from crosses involving the translocation. The mutant form of *co* is undoubtedly present since typically *co* spores were recovered, but the phenotype of the strains themselves was clearly not typically *co*. Examination of spores from crosses of the pseudo-wilds to *co* showed that although they were quite possibly pure with respect to *pyr* they were not pure with respect to *co*. Spores were plated on minimal and on minimal supplemented with cytidine. Those which were phenotypically wild, *pyr* and *co* on these two media are recorded below.

	MINIMAL			CYTIDINE		
	WILD	*pyr*	*co*	WILD	*co*	% WILD
co A × 9 R 2 a	8	873	864	136	1627	8
co A × 9 R 4 a	72	2002	1857	510	921	36
co a × 9 R 6 A	16	1035	980	409	2695	13
co A × 9 R 7 a	30	931	830	1044	1491	41
co a × 9 R 8 A	21	1370	1482	630	1074	37
co A × 9 R 9 a	1	1173	1216	52	1329	4

From two of these crosses 69 asci from 13 perithecia were dissected and the spores were allowed to germinate on minimal with cytidine. The asci were found to contain either eight *co* spores of four *co* and four not-*co* spores, but in no case were both types of asci obtained from the same perithecium.

These results suggest that each of these pseudo-wild strains contains at least two types of nuclei, of the constitutions, *co pyr* and + *pyr*, and that, in each perithecium, one or the other of these types functioned as the parent nucleus contributed by the pseudo-wild. From the crosses to wild, spores which were *co* + could be attributed to cross-overs in zygote nuclei of the

constitution, *co pyr* / ++. From crosses to *co*, spores which were phenotypically wild on minimal medium could result from crossing-over in *co* + / + *pyr* zygote nuclei. Some of the latter spores may have been pseudo-wild, however, as will appear below. Since both *co pyr* and + *pyr* were recovered, it seems most probable that both *pyr-3a* and *pyr-3d* were recovered from the same strain.

The cross of *co pyr-3d* to *arg* was made to obtain the triple mutant so that *arg* might be introduced as a second marker in crosses of *pyr-3a* to *pyr-3d*. The small percentage of wilds observed suggested that *arg* may be located between *co* and *pyr* and that, since wilds from crossing-over would then be expected to be very infrequent, the wilds observed might, therefore, be pseudo-wilds. Two of these, when transferred to Westergaard's minimal, showed the dilute "colonial" character, as had pseudo-wilds from *co pyr-3d* × *pyr-3a*. The two strains were crossed to standard wilds, 1400-4A and 1347-2a, and spores were plated on three media, minimal, minimal with cytidine and minimal with arginine. The spores were thus classified and counted as follows:

	MEDIUM	WILD	*pyr-3d* AND/OR *arg*	*co*
Wild A × 11 R 1 a	minimal	1062	1050 = 50%	0
Wild A × 11 R 1 a	cytidine	1269	1208 = 48%	29
Wild A × 11 R 1 a	arginine	1511	66 = 4%	2
Wild a × 11 R 2 A	minimal	588	557 = 48%	3
Wild a × 11 R 2 A	cytidine	1006	368 = 19%	527
Wild a × 11 R 2 A	arginine	992	372 = 27%	8

From this it appears that both + *arg* + and *co* + *pyr-3d* were recovered from each strain. This was confirmed by isolating 20 mutant-appearing spores from each minimal plate, culturing them on minimal with cytidine and arginine and testing them for their response to these compounds. From the 11 R 1 cross, two responded only to cytidine and 18 only to arginine and from the 11 R 2 cross 10 responded to cytidine and 10 to arginine. Those which responded to cytidine were *co;* word those which responded to arginine were not-*co*.

Pseudo-wilds from co × *pyr-3d T.*—From this cross five spores which appeared phenotypically wild on minimal plates were isolated and cultured on Westergaard's minimal. Four of these remained phenotypically wild in slant culture, but the fifth, 2 R 8, became distinguishable from *co*. In flasks, after 4 days growth at 25°C., they gave the following dry weights, in mg.: 2 R 6—52; 2 R 7—56; 2 R 8—40; 2 R 9—52; 2 R 10—52. When crossed to wild each strain gave fertile perithecia with one mating type and no reaction with the other. Spore counts from plates of minimal and minimal with cytidine are given below.

	MINIMAL			CYTIDINE	
	WILD	*pyr*	*co*	WILD	*co*
Wild a × 2 R 6 A	814	824	0	927	0
Wild a × 2 R 7 A	576	488	0	635	0
Wild A × 2 R 8 a	726	0	478	...	...
Wild a × 2 R 9 A	302	281	0	654	0
Wild a × 2 R 10 A	588	598	0	813	0

These crosses, including the one involving 2 R 8 showed the type of sterility associated with the translocation. Since 2 R 8 was not phenotypically *co* on the agar plate, but later showed this character, it seems possible that it was, at least originally, pseudo-wild. These strains resemble pseudo-wilds from the *pyr-3d* T × *pyr-3b* cross in that one or the other but not both parental types were recovered.

Asci Containing Pseudo-wilds.—From the *co* × *pyr-3d* T cross asci which contained part normal and part defective spores were examined in an attempt to learn whether pseudo-wilds came from these. (Aberrant segregations of *pyr* have not been observed in asci with 8 normal spores.) Of 13 asci which had two normal spore pairs in one half of the ascus and two defective pairs in the other, four contained pseudo-wilds. From two of these asci (1296 and 1320) both normal pairs were pseudo-wild. From the other two (1358 and 1360) one pair was pseudo-wild and the other was *co* +. The remaining nine asci of this type contained one + *pyr* pair and one *co* + pair. Asci of this type, from mature perithecia, were rather infrequent;[8] it was usually necessary to examine several perithecia in order to find one. Another type of ascus which is very frequent contains one normal and one aborted pair in each half. Only eight of this type were examined and no pseudo-wilds were found. The pseudo-wilds isolated were not tested in flasks, but they behaved as wild when tested in small tubes of liquid minimal. The six strains remained phenotypically not-*co* in slant cultures and *pyr* was recovered from crosses of each one to standard wild. Spores from crosses involving 1320-1 and -2 were not counted but counts from the four other crosses appear below.

	WILD	*pyr*	*co*	% *pyr*
Wild a × 1296-1A	609	489	0	44
Wild A × 1296-2a	685	541	0	44
Wild a × 1358-1A	1278	964	0	43
Wild a × 1360-4A	1242	945	0	43

Asci from each of these crosses showed patterned spore abortion but this was not true of those from crosses of the phenotypically *co* + strains 1358-2A and 1360-3A to wild. Spores from the latter crosses were plated but not counted. Pyrimidine mutant spores were not observed.

Studies of asci containing normal and defective spores, from crosses not involving the translocation, are in progress. Asci of this type are more

difficult to find from these crosses, which may reflect the fact that phenotypically wild progeny were less frequent, at least among germinated spores. One ascus of the same type as 1296 was among those dissected from a cross to *co* of pseudo-wild 9 R 4 (from *pyr-3a* × *co pyr-3d*). Other asci from the same perithecium contained both *co* + and + *pyr* spores, hence, presumably, the parent nuclei were of these constitutions. From this ascus one member of each of the two normal pairs germinated; both appeared phenotypically wild on the plate but subsequently showed the dilute "colonial" character. One strain grew very slowly on minimal and was finally discarded. The other was crossed to *co* and a few asci were dissected. Five asci from one perithecium contained only *co* + spores but 6 asci from 3 other perithecia contained both *co* + and + *pyr* spores. From *pyr-3a* × *co pyr-3d* an ascus containing one pseudo-wild spore pair was found. The spores of the other pair occupying the same half of the ascus were defective; the four spores in the other half appeared normal but failed to germinate. These observations suggest the possibility that, whether the translocation is present or absent, spores which give rise to pseudo-wilds occur in asci which also contain defective spores.

Discussion.—The occurrence of pseudo-wild serves to emphasize the questionable nature of the conclusion, based solely on the appearance of phenotypically wild progeny, that crossing-over between two mutant genes has taken place. Studies which will be reported later, on other mutants, have shown that the ability to produce pseudo-wilds is not confined to pyrimidine mutants or to mutants in the *pyr* linkage group. Hence, it seems desirable that this phenomenon be considered in attempting to evaluate data from crosses between Neurospora mutants which are closely linked or possibly allelic.

A satisfactory explanation of the origin of pseudo-wilds must account for their growth on minimal medium, for the types of progeny recovered from out-crosses, for the difference in their behavior depending upon the presence of the translocation in the parent cross, and for the development of the dilute "colonial" phenotype. Tentative explanations which have been considered are as follows: that suppression of the mutant character is effected by chance combinations of a number of genes; that back-mutation at a *pyr* locus results in the formation of a pyrimidine-independent heterocaryon in which *pyr* mutant nuclei predominate; that $n + 1$ nuclei, which arise during meiosis as a result of non-disjunction of the *pyr* chromosome pair, account for pyrimidine-independence but are not usually transmitted in crosses; that a heterocaryon, capable of wild-type growth, is established by the inclusion of genetically different nuclei in an ascospore.

The behavior of pseudo-wilds from crosses involving the translocation may be explained in terms of back mutation of *pyr* and, possibly, of *co* in the case of 2 R 8, if it is supposed that, for some reason, nuclei carrying

the back-mutation remain greatly in the minority. Suppression of the mutant character by a chance combination of several genes also seems an adequate interpretation of these cases except that it does not account for the change in phenotype of 2 R 8, from not-*co* to *co* unless, perhaps, mutation of a gene of the suppressor complex is assumed. The origin of these strains from heterocaryotic spores seems less probable because of the lack of evidence from out-crosses that pseudo-wilds from *pyr-3b* × *pyr-3d* T and *co* × *pyr-3d* T were hetercaryotic. From the latter cross a pyrimidine-independent heterocaryon from which *pyr* could be recovered would presumably also contain nuclei carrying the normal allele of *pyr* but such nuclei would, most commonly, carry the mutant allele of *co*. Since, when *pyr* was recovered, *co* was not recovered, it would be necessary to suppose that back-mutation of *co* or crossing-over between *co* and *pyr* accompanied the formation of a heterocaryotic spore in each case studied, or that *co* + nuclei were present but so infrequent that no *co* + spores were found among the progeny of the eight crosses. Also, the types of asci from which the strains have been obtained are not those which would be expected to contain heterocaryotic spores. Since the nucleus functions in cutting out the spore[4, 10] there would seem to be no reason for the appearance of the full complement of eight spores if two nuclei were included in each of two or four spores. If additional nuclear divisions prior to spore formation are invoked to remove this difficulty there would appear no reason for two or four of the eight spores to be defective.

With regard to pseudo-wilds from *co pyr-3d* × *pyr-3a* and *co pyr-3d* × *arg*, back-mutation seems an improbable explanation, since, in the first case, two, and in the second, three, back-mutations per strain would be required. Because of the recovery of both parental types from the same strain the action of multiple-gene suppressors need not be considered. Derivation from heterocaryotic spores, on the other hand, remains a possible explanation since these strains behaved as if they were heterocaryotic and, at least in connection with those involving *arg*, there is no reason to doubt that a heterocaryon thus established would be capable of wild type growth on unsupplemented medium.

The explanation based on the origin of pseudo-wilds from ascospores with $n + 1$ nuclei can be made to appear consistent with the behavior of both types of pseudo-wilds if, in addition to the assumptions that such nuclei account for pyrimidine-independence but do not usually function in crosses, a further assumption is made. This is that in mitotic divisions $n + 1$ nuclei give rise to haploid nuclei by some mechanism such as somatic non-disjunction[11] and that it is the haploid nuclei which usually function in out-crosses. It may be pointed out that there are available no means of evaluating the justification for these suppositions. They can, however, be used as a basis on which to account for the fact that pseudo-wilds from

crosses involving the translocation gave, among progeny from out-crosses, only one parental type, whereas those from crosses without the translocation gave both. For this purpose it may be supposed that, in isolates tested from *pyr-3d* × *pyr-3d* T and *co* × *pyr-3d* T, the original disomic nucleus contained one normal and one translocated member of the *pyr* chromosome pair, and that, in the first case, *pyr-3b* and *pyr-3d*, and in the second, *co* + and + *pyr-3d* were thus both present in the same nucleus. If, for example a translocated chromatid from the other chromosome pair (linkage group B) involved in the translocation were included in the disomic nucleus, then a haploid nucleus which arose from it would be viable if it contained the translocated members of the *pyr* pair, but inviable if it contained the corresponding normal chromosome. Hence, if disomic nuclei did not function in the out-cross, only the mutant gene carried by the translocated *pyr* chromosome would appear among the progeny. Which mutant appeared in a given case would depend upon whether recombination of the mutant genes and the translocation occurred as a result of crossing-over and upon the way in which the chromatids of the B chromosome pair segregated. In the absence of the translocation there would be no such restriction on the type of viable haploid nuclei which could be formed and both members of the *pyr* pair could, therefore, be recovered from one strain. The process of formation of hapolid nuclei could serve also to explain the change in phenotype, with respect to *co*, of 2 R 8 and of the pseudo-wilds from crosses of *co pyr-3d* to *pyr-3a* and *arg*. It would, of course, be necessary to suppose that, at least in pseudo-wilds from which only one *pyr* mutant type was recovered, disomic nuclei persist in vegetative culture and that the pseudo-wilds tested are heterocaryons containing disomic as well as haploid nuclei. This explanation is perhaps the more convenient one of those suggested but the authors would like to emphasize that there is no direct evidence to support it and that it can probably be established with finality only by the cytological demonstration of the presence of disomic nuclei.

Even if the occurrence of $n + 1$ nuclei or of heterocaryotic spores were established as the explanation of the pseudo-wilds, the question of the genetic relationship between *pyr-3a* and *pyr-3d* would remain unanswered. The possibilities that they represent mutations at different loci or different mutations at the same locus would have to be considered equally probable since no evidence exists that in either case the presence of both *pyr-3a* and *pyr-3d* in the same nucleus would not establish a wild phenotype. The same, of course, applies if they were present together in a heterocaryon. From a practical standpoint, then, it seems useless at present to consider the question of whether the mutants are allelic. However, the fact that phenotypically wild strains have been obtained from crosses of *pyr-3d* to *pyr-3b* but have not yet been observed from crosses of *pyr-3a* to *pyr-3b* suggests the possibility that *pyr-3a* and *-3d* differ in some way. It may

be possible, by studying strains from asci with both normal and defective spores, to show that the cross of *pyr-3a* to *pyr-3b* yields strains which are not phenotypically wild but from which both parent mutants can be recovered. If so, investigation in this manner of other *pyr* mutants which are phenotypically similar to *pyr-3a* and *-3d* may prove to be of interest in showing how many types there are with respect to the ability to produce, in intercrosses, strains of this kind which are phenotypically wild.

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THE GENOTYPE OF THE ENDOSPERM AND EMBRYO AS IT INFLUENCES VIVIPARY IN MAIZE

BY DONALD S. ROBERTSON†

CALIFORNIA INSTITUTE OF TECHNOLOGY* AND THE BIBLE INSTITUTE OF LOS ANGELES

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The development of the maize seed is dependent on the orderly unfolding of events in which each component of the developing caryopsis has a particular role to play. The ultimate control of these events must depend upon numerous genes, which if altered will interfere with normal development. Many mutants of this type have been described, ranging from those which produce relatively slight alteration in the caryopsis to those which prevent practically all development. Among those producing relatively slight changes are mutants which give rise to premature germination. The seeds of these mutants develop normally until late in ontogeny. During the early dough stage the plumule begins to elongate, and the seeds germinate while still attached to the ear. Such mutants have been called viviparous.

The viviparous gene may induce changes in one or more of the various tissues of the developing caryopsis. Since viviparous seeds segregate on the ear, any changes induced in maternal tissue cannot determine whether a seed will be viviparous. Changes responsible for vivipary must be produced in tissues resulting from the union of male and female gametes—the embryo and/or the endosperm. There are three possible ways that the viviparous gene can promote premature germination: (1) by inducing in the embryo changes which allow it to germinate under conditions normally not conducive to germination; (2) by inducing in the endosperm changes which alter the normal germination inhibiting mechanism; and (3) by inducing changes in both the endosperm and embryo which interact to overcome the normal germination inhibition. In order to determine which of these three possibilities actually is operative within the mutant seed, it is first necessary to isolate the effect produced by the viviparous genotype of the endosperm from that of the embryo.

Seeds resulting from hetero-fertilization frequently have embryos and endosperms of different genetic constitution. Such seeds are produced when the egg and polar nuclei of the egg sac fuse with sperm nuclei of different

TABLE 1

	GENOTYPE OF THE ENDOSPERM	GENOTYPE OF THE EMBRYO
Class A	*Vp Vp vp vp*	*vp*
Class B	*vp vp*	*Vp Vp vp*

pollen grains. Sprague[1] observed a few such seeds on an ear segregating for one of the viviparous mutants. He found that only the seeds with genetically viviparous embryos germinated. This would indicate that in this mutant, at least, the gene effects changes in the embryo which induce germination.

The use of A-B translocations makes possible the systematic production of embryos and endosperms of differing genetic constitution. In some strains of maize there may be present, besides the basic set of ten A-type chromosomes, one or more supernumerary, genetically inert, chromosomes which have been designated as B-type. Roman[2] has described the behavior of A-B interchanges in the production of the male gametes. In the division of the generative nucleus of the developing pollen grain the B centromeres usually fail to separate, giving one sperm both B centromeres and the other none. Where a portion of an A-type chromosome is attached to the B centromere, this non-disjunction produces within a given pollen grain two sperm of different genetic constitution. One sperm has the portion of the chromosome attached to the B centromere present in duplicate (hyperploid sperm), whereas the other is deficient for this region (deficient sperm). If the viviparous gene is located in the trans-

located section of the A chromosome, two classes of seeds are obtained from pollination of viviparous by an A-B interchange. These two classes are shown in table 1. Class A seeds are the result of the hyperploid sperm fusing with the polar fusion nucleus, and the deficient sperm fusing with the egg nucleus; whereas Class B seeds are the result of the reciprocal fusion. In both classes the endosperm and embryo differ with reference to their viviparous genotype.

Although these classes are theoretically possible, they are not readily recognized by the expression of vivipary alone. If one of these classes of seeds were to germinate prematurely, there is no way to determine *a priori* to which class it belongs. However, it is possible to distinguish these classes in some viviparous mutants which interrupt the normal carotenoid and chlorophyll development. Seeds of these mutants have white or pale yellow endosperms and germinate prematurely, producing albino seedlings. By using such a mutant in combination with the A-B interchanges, it is possible to recognize the two types of seeds shown in table 1. Class A seeds should have yellow endosperms because they carry the normal alleles of vivipary and produce white seedlings, since only the mutant allele is present in the embryo. The Class B seeds would be recognizable by white endosperms and green seedlings.[3]

Plants heterozygous for viviparous-5, a mutant which inhibits carotenoid and chorophyll development as well as inducing premature germination, was pollinated by pollen of plants carrying A-B interchange TB-1b. The resulting ears were found to be segregating viviparous and white seeds. A classification of endosprem and seedling color revealed three types of seeds: (1) yellow endosprem with albino seedlings, (2) white endosperm with green seedlings, and (3) yellow endosperm with green seedlings. Since viviparous-5 affects carotenoid formation, it is possible to recognize the first type of seeds as those belonging to Class A. The second type of seeds are those belonging to Class B. The third type results from the fertilization of ovules carrying the normal alleles of viviparous-5 and are, therefore, not important in this consideration. Thus, the two classes of seeds shown in table 1 are recognizable. Only seeds of Class A were found to be viviparous, whereas those of Class B were dormant. As is expected, all seeds of Class B grown to maturity and self-pollinated have segregated for viviparous-5.

By using TB-1b in combination with viviparous-5, it has been possible to distinguish between the role of the endosperm and that of the embryo in the production of premature germinating seeds. The data suggest that the endosperm does not play an active role in vivipary, since seeds germinate prematurely if they have a normal or mutant endosperm as long as the embryo is genetically viviparous. Thus, it would appear that vivipary is determined by the genotype of the embryo and is independent of the

genotype of the endosperm. The evidence from hetero-fertilization supports this conclusion.

The nature of the mechanism responsible for the dormancy of the developing embryo is not known. Work of La Rue,[4, 5] Haagen-Smit *et al.*,[6] and others has shown that normal corn embryos are capable of germination soon after pollination, if removed from the developing caryopsis and cultured. Thus, the developmental dormancy is not entirely dependent upon the embryo but is influenced by some environmental factor within the developing seed. The genetic evidence and that from embryo culture experiments suggest that viviparous seeds germinate prematurely because their embryos are capable of germination within the caryopsis in an environment which will not permit the germination of normal embryos.

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† Permanent address, The Bible Institute of Los Angeles, 558 South Hope Street, Los Angeles, Calif.

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*SUBSTRATE STABILIZATION OF ENZYME-FORMING CAPACITY DURING THE SEGREGATION OF A HETEROZYGOTE**

BY S. SPIEGELMAN AND W. F. DELORENZO†

DEPARTMENT OF BACTERIOLOGY, UNIVERSITY OF ILLINOIS, URBANA, ILLINOIS

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Introduction.—In the course of investigating the inheritance of the capacity to synthesize "galactozymase" Winge and Roberts[1] discovered a "slow" adapting phenotype. These authors demonstrated that the character was determined by a single recessive allele (g_s) which segregates normally in crosses to the wild type bearing the dominant (G).

Analysis by Spiegelman, Sussman and Pinska[2] of g_s-genotype cultures revealed the existence of heritable differences in phenotype among cells carrying this allele. This heterogeneity is easily and simply exhibited by plating glucose-grown g_s-cells on purified galactose agar test plates upon

which two kind of clones develop. The predominant type is small and negative in appearance. Present in a proportion of about one in a thousand, however, are cells which yield large positive colonies. Such positives retain and transmit enzyme-forming capacity for an indefinite number of generations provided galactose represents the sole or primary source of carbon and energy. If, however, positive cells are allowed to grow in the absence of this substrate, a mass reversion to the original negative phenotype occurs quite abruptly between the 6th and 7th generations.

The data obtained led to the conclusion that the inheritance of the capacity to synthesize galactozymase in this strain is a predominantly cytoplasmic event and involves the discrete transmission of elements necessary for enzyme formation. On the basis of the behavior of positive cells in the presence and absence of substrate it was concluded these elements can increase in the presence of galactose but not in its absence. The reversion of a positive culture to the negative phenotype on growth in the absence of substrate was thus explained in terms of the dilution of the enzyme-forming elements to a level at which cells are produced possessing either none or an insufficient number of these elements.

These facts and conclusions received direct support from subsequent experiments by Spiegelman, DeLorenzo and Campbell[3] in which the single progeny cells produced by a positive during division in the absence of galactose were serially isolated and characterized. Starting with the 5th bud, cells of the negative phenotype were produced with increasing frequency. Thus, e.g., only 33% of the 8th buds produced were of the positive phenotype.

The data obtained in the course of these single-cell experiments permitted a more detailed analysis of the reversion phenomenon than was possible from the earlier mass population studies. The results are accurately described by the following quantitative relations: (*a*) a fully adapted positive cell contains in the neighborhood of 100 particles; (*b*) the minimal number required for the positive phenotype is in the neighborhood of 1; (*c*) the probability of any given element passing into the daughter cell in the course of a division is close to one-half.

These findings lead one to inquire why discrete cytoplasmic transmission of enzyme-forming capacity is easily exhibited in cells of the g_s-genotype whereas this does not obtain in those carrying the dominant allele. All cells of the *G*-genotype, adapted and unadapted, yield positive clones on the test plates. In terms of our previous findings the following two alternatives may be considered to explain the difference between the two genotypes: (*a*) the mechanism of galactozymase formation in *G*-genotype cells is fundamentally different and does not involve particulate cytoplasmic elements analogous or related to those detectable in the g_s-genotype; (*b*) the mechanism of enzyme synthesis in the two genotypes is the

same and the difference resides in either a greater stability of the *G*-genotype elements or in a more efficient production of them, so that every *G*-genotype cell has one or more of these elements at all times. The latter hypothesis would presume, in analogy with the findings with g_s-cells, that adapted *G*-cells would contain more of these elements than unadapted ones.

Information on such issues can in principle be obtained by comparing the effects of introducing into g_s-cells adapted and unadapted cytoplasm derived from cells possessing the dominant allele. On the basis of the first hypothesis little or no effect on the g_s-phenotype would be expected *a priori* from either type of cytoplasm. On the other hand if the second alternative obtains, the introduction of adapted cytoplasm should convert g_s-genotype cells to the positive phenotype and do so more effectively than cytoplasm from unadapted *G*-cells. In addition it would be expected that the acquired positive phenotype would be indefinitely transmitted by the g_s-cells in the presence of substrate.

It is the purpose of the present paper to describe the results of such experiments. The method adopted for attaining the desired experimental situation is identical to that employed in a previous[4] investigation of the effect of melibiose on the segregation of melibiozymase-forming capacity.

The procedure is based on the fact that in ascus formation each of the four haploid segregant spores derives its cytoplasm from the diploid. Thus, with the aid of the adaptable diploid heterozygote Gg_s, adapted or unadapted cytoplasm can be introduced into cells of the g_s-genotype depending on the treatment of the diploid prior to segregation of the haploid spores. The results obtained are in agreement with those observed with the melibiozymase system.

Methods and Materials.—Strain 55, a diploid derived from a pedigree involving hybridizations and backcrosses between *S. cerevisiae* and *S. chevalieri*, was employed. It was obtained through the generosity of Professor O. Winge. Strain 55 has the genetic constitution (*Gg*$_s$ *Mm Dd*) being heterozygous for galactose and maltose fermentation as well as the diploidizing gene.[5]

The media, carbohydrates and methods of growth were the same as those used in previous[2,3] studies of the "slow" adaptation phenomenon.

The induction of ascus formation was achieved by the use of Adams'[6] presporulation and sporulation media which possess the advantage that control of carbohydrate composition is more effectively attained than with other methods. Strain 55 takes 4 days to show evidence of sporulation on this medium. To minimize the confusion which Winge and Roberts[7] pointed out can result from multiple cycles of sporulation and recombination, sporulating slants older than 6 days were not employed.

Dissections were carried out as previously[3] described. The isolated spores were transferred separately by means of a micropipette to a drop

of 2% galactose-0.075% glucose broth held on a coverslip which was then placed on a Van Tiegham chamber.

After 24–48 hrs. at 30°C. each spore can yield micro-clones of between 500 to 1000 cells which are then transferred and spread on Eosine Methylene Blue-purified galactose (EMB-PG) agar test plates. A spore is scored as positive if all the cells of the microclone derived from it yield positive colonies on the test plates. A spore is scored as negative if negative colonies are produced under the same conditions.

Experimental Results.—To subject the issues raised in the introduction to experimental analysis a comparison was made of the segregation of the galactose phenotype under the following two conditions: (1) Control dissections were carried out with unadapted glucose grown cells of strain 55 which were sporulated in the absence of galactose. The resulting asci were dissected and the individual spores allowed to produce microclones which were then plated and scored; (2) in the experimental set, cells of strain 55 were adapted prior to sporulation by 24-hr. growth in galactose. Contact with substrate was then maintained throughout all subsequent steps of sporulation and ascus dissection. It will be noted that all micro-clones in this series of experiments were grown in the presence of galactose. However, in the control procedure the cells do not come in contact with substrate until after segregation has occurred.

Control and experimental series of dissections were carried out in parallel. A set of such dissections is detailed in table 1, which illustrates the methodology of these experiments. All eight control asci in this series yielded the 2+:2− ratio expected of a heterozygote. On the other hand, in the experimental series all of the asci showed excess positives, 7 yielding 4+:0− and one 3+:1−.

To examine further the nature of the excess positives observed in the experimentals and compare them with the positives which appear in the control segregations, all positives in the present investigation were put through the following reversion growth test in the absence of substrate. Two clones were chosen at random from each positive test plate, seeded into separate tubes containing 2% glucose broth, and allowed to go through about 15 generations. They were then replated on EMB-PG test plates and scored. Those positive spore clones that reverted to the negative phenotype are indicated with an asterisk in table 1. None of the positives derived from this set of control segregations reverted. In the experimentals, however, two out of every 4+:0− ascus and one of the three in the 3+1− ascus reverted to the negative phenotype, thus restoring the Mendelian ratio. Thus the reversion test clearly reveals the non-Mendelian nature of the excess positives observed in the segregation of the adapted heterozygotes.

Table 2 summarizes the results obtained in the dissection of 68 asci in

TABLE 1

THE EFFECT OF SUBSTRATE ON THE SEGREGATION OF ENZYME FORMING CAPACITY IN A *Gg*$_s$ HETEROZYGOTE

(+) indicates that the microclone (*ca.* 500 cells) developed from the corresponding spore yielded uniformly positive colonies on the test plate. (−) indicates that the negative phenotype was observed in a comparable test. All positives in both the control and experimental series were put through a reversion test growth in the absence of substrate. An asterisk indicates that the corresponding clone reverted to the negative phenotype.

EXPERIMENTALS IN PRESENCE OF SUBSTRATE					CONTROLS IN ABSENCE OF SUBSTRATE				
ASCUS NO.	SPORES A	B	C	D	ASCUS NO.	SPORES A	B	C	D
13	+*	+	+*	+	47	−	+	−	+
14	+*	+	+	+*	48	−	−	+	+
15	+*	+*	+	+	50	+	−	+	−
17	+	+*	+	+*	51	+	−	+	−
20	+	+*	+	+*	52	−	+	+	−
21	+	+*	+*	+	53	−	+	−	+
23	+	+*	+*	+	56	−	+	−	+
29	+*	+	+	−	58	+	+	−	−

TABLE 2

SUMMARY OF ALL FOUR SPORED ASCI RECOVERED WHICH WERE SEGREGATED IN PRESENCE AND ABSENCE OF SUBSTRATE

ASCUS TYPES		NUMBER FOUND	
BEFORE REVERSION +:−	AFTER REVERSION +:−	IN ABSENCE OF SUBSTRATE	IN PRESENCE OF SUBSTRATE
2:2	2:2	34	1
4:0	2:2	0	22
4:0	4:0	2	2
0:4	0:4	1	0
3:1	2:2	1	5

TABLE 3

SUMMARY OF SPORE PHENOTYPES BEFORE AND AFTER REVERSION GROWTHS IN EXPERIMENTAL AND CONTROL SERIES

Control spores derived from unadapted heterozygotes; experimental spores derived from adapted heterozygotes segregating in the presence of substrate. *P* indicates the predicted frequency of the observed deviations from the expected as determined by the Chi Square method.

CONDITION		BEFORE REVERSION GROWTH +:−	AFTER REVERSION GROWTH +:−
Control	Found	233:237	227:243
	Expected	235:235	235:235
	P	0.86	0.62
Experimental	Found	175:13	98:90
	Expected	94:94	94:94
	P	$<1 \times 10^{-9}$	0.58

which all 4 spores were recovered, scored and tested for reversion. Thirty-four out of 38 control dissections yielded a ratio of 2+ : 2−. Three genetically exceptional control asci were observed, two being 4+ : 0−, none of which reverted, and one being 0+ : 4−. These exceptional asci were further analyzed for the other two characters, maltose fermentation and the presence of the *D* gene. They were found to be homozygous for these markers as well as the galactose character and had presumably therefore resulted from self-diploidization.

In the experimental series it will be noted that only one out of 28 segregations in the presence of substrate resulted in a 2+ : 2− ratio. Twenty-four were 4+ : 0−, and five were 3+ : 1− on the original scoring for phenotype. All but two reverted to the 2+ : 2− ratio on growth of the positives in the absence of substrate. The two exceptional asci proved on subsequent analysis to be homozygous for all markers and may be disregarded as self-diploids.

TABLE 4

EFFECT OF SUBSTRATE ON SEGREGATION OF ENZYME-FORMING CAPACITY IN A $g_s g_s$ HOMOZYGOTE

All diploid $g_s g_s$ cells and their segregants were treated with substrate in a manner identical to that described for the experimental series of heterozygote (Gg_s).

NUMBER OF SPORES RECOVERED PER ASCUS	NUMBER OF ASCI	PHENOTYPE +:−
4	26	0:4
3	33	0:3
2	26	0:2
1	9	0:1
Total Number of Spores	264	0:264

Thus far we have discussed the results obtained with asci in which all four spores were recovered. It is evident from the results obtained that the ability of substrate to modify phenotype segregation ratios should be easily detectable on the basis of single spore characterization without reference to the analysis of tetrads. That this is so may be seen from table 3 in which are summarized the results from the dissection of 218 asci. In the controls a ratio of 233+ : 237− was observed whereas in the experimentals an initial ratio of 175+ : 13− was obtained which on a reversion growth became 98+ : 90−.

Control experiments were run to test whether the experimental procedure could convert g_s cells to the positive phenotype in the absence of the dominant *G*-gene. This was accomplished with the aid of a $g_s g_s$ homozygote derived from a segregant of strain 55. This diploid was put through the entire experimental treatment and thus experienced contact with galactose equivalent in time and extent to that of the Gg_s heterozygotes in

the experimental series. The results obtained with the homozygote are summarized in table 4. Out of 264 spores recovered and scored, not a single one exhibited the positive phenotype. This is in sharp contrast to the results obtained with the heterozygote and leads to the conclusion that the presence of the dominant allele is a necessary component in the mechanism of the phenomenon observed.

Discussion.—The data demonstrate that prior adaptation to galactose, and its continued presence during sporulation, can modify the ratios of the corresponding enzymatic phenotype observed during the segregation of a Gg_s heterozygote. These results are in agreement with an earlier report by Spiegelman, *et al.*,[4] in which it was found that melibiose exerted a similar influence on the segregation of melibiozymase forming capacity in a diploid strain heterozygous for this character. Positive spores, yielding clones capable of fermenting melibiose, were produced far in excess of the expected 2+:2− ratio which characterized the controls. As in the present instance, de-adaptation led to the restitution of the normal Mendelian ratio of the phenotypes. A cytoplasmic mechanism was invoked to explain these findings.

Winge and Roberts[1] in summarizing their view of the nature of long-term adaptation state that it "is an expression of the fact that enzyme production by the yeast in the presence of the sugar in question is gradually increased until it exists in sufficiently high concentration to bring about visible fermentation." These authors quite correctly emphasized that from the viewpoint of final outcome it was not possible to distinguish the results of the melibiose experiments cited above from their own findings with galactozymase. In view of their interpretation of the "slow" genotype it appeared no longer necessary to invoke cytoplasmic transmission to explain such substrate induced abnormal segregations of phenotype. The pretreatment of the heterozygote with galactose and the subsequent experimental steps are such that the period of contact with substrate prior to dissection and scoring is a relatively extensive one. It is conceivable that it might be sufficient in itself to convert g_s-genotype cells into positives. However, the experiments reported here make this interpretation untenable. Adapted heterozygotes *do* yield galactose positive spores of the g_s-genotype under conditions in which homozygous g_sg_s diploids *do not* yield phenotypically positive spores.

It seems necessary to conclude that the presence in a diploid cell of the dominant allele and substrate lead to the accumulation of elements, which if incorporated into the cytoplasm of a g_s-spore, confer upon it the ability to give rise to progeny able to synthesize enzyme. This acquired capacity is indefinitely transmissible in the presence of substrate but is lost on growth in its absence.

Adapted cytoplasm of Gg_s cells is clearly more effective in converting

g_s-spores to the positive phenotype. Consequently, enzyme formation in cells containing the dominant gene appears to involve an increase in cytoplasmic components which are at least sufficiently related to the galactozymase synthesizing mechanism of the g_s-genotype as to be effective in initiating its activity. This finding makes unlikely explanations of the difference between the two genotypes which postulate a fundamentally different mechanism of galactozymase formation.

Since all cells containing the dominant yield positive clones on the galactose test plates, whereas only one in a thousand g_s-cells does so, it is evident that the mechanisms of galactozymase synthesis in G- and g_s-cells, though related, are clearly not identical. Within the framework of the findings thus far discussed, it seems of interest to see whether the difference in behavior might not, in part, be due to the fact that G-cells can produce the necessary cytoplasmic elements even in the absence of substrate.

A possibility of this nature can in principle be detected by performing a large number of dissections of unadapted heterozygotes analogous to the controls of the experiments detailed above. This procedure essentially uses g_s-spores to sample unadapted Gg_s cytoplasm for the presence of the relevant active elements. If these units can be produced in the absence of substrate, aberrant g_s-positives should be obtained in such dissections with a frequency higher than the spontaneous background rate of one in a thousand.

In the course of the present experiments a rather large series (190 asci) of control dissections were included in an attempt to obtain information relevant to this issue. The results are summarized in table 3. It will be noted that 6 out of the 243 g_s-genotype spores recovered from the control asci were phenotypically positive on the original test and reverted to the negative phenotype after several (7 to 12) divisions in the absence of substrate. This frequency of 2.5% is considerably higher than the spontaneous rate of 0.1% for g_s-cells. Furthermore it will be noted from table 4 that none of the 264 spores obtained from the g_sg_s homozygote exhibited the positive phenotype. These results suggest therefore that even unadapted Gg_s-cells contain some of the cytoplasmic units which can convert g_s-spores to the positive phenotype and support the view that these units can be formed in the absence of substrate.

It is at present impossible to provide definitive information concerning the nature of the active elements derivable from G-cells and capable of converting g_s-cells to the positive phenotype. Neither can anything precise be said concerning the possible relation of these units to those which are formed spontaneously but rarely in cells of the g_s-genotype. The results obtained thus far on the factors controlling the transmission of these fermentative enzymes parallel in many ways the important con-

tributions of Ephrussi[8] and his collaborators on the inheritance of the respiratory enzyme system in yeast. Whether this parallelism of the two investigations extends beyond that of superficial similarity can be decided only by further research.

Winge[9] has recently accepted explicitly a cytoplasmic interpretation of long-term adaptation. He adopts the view previously proposed by Spiegelman, *et al.*,[4] to explain the melibiose experiments. In this hypothesis it is presumed that the cytoplasmic elements involved are the enzyme molecules themselves which are capable of autocatalytic increase. However, no conclusive data are available which establish the validity of this assertion. In view of the fact that a system is now available which permits an experimental analysis of the question, it would seem advisable to avoid categorical identification of these elements with the enzyme molecules. Under the circumstances such neutral terms as "enzyme-forming elements" or "cytoplasmic units" would appear to be preferable, since they leave open for future experimental decision the question of their identity. In connection with this problem, it should be noted that the analysis[3] of the reversion phenomenon suggests that the presence of one cytoplasmic element is sufficient for a cell to exhibit the positive phenotype on a test plate. This finding clearly increases considerably the difficulty of designing a decisive experiment which seeks to either confirm or deny the identification of the relevant element with an enzyme molecule.

Summary.—The effect of prior adaptation of heterozygotes to galactose on the segregation of the capacity to form galactozymase in yeast has been studied. Unadapted heterozygotes of Winge's strain 55, with few exceptions, yield 1:1 ratio of positives to negatives on galactose test plates. This result agrees with the previous report of Winge and Roberts.[1] However, when the heterozygote is previously adapted and the segregation occurs in the presence of substrate, all four segregant spores exhibit the positive phenotype in over 90% of the asci dissected. Growth for 7 to 12 divisions of four such positive spore-clones leads to a reversion of two of them to the negative phenotype, thus restoring the Mendelian ratio of phenotype. Homozygous recessives carried through the same procedure do not yield positive spores.

These data are explained in terms of cytoplasmic elements necessary for enzyme formation and produced in the presence of substrate and the dominant gene, *G*. When these elements are incorporated into the cytoplasm of the g_s-spores during segregation they are converted to the positive phenotype. The abrupt reversion of such positive cultures to the negative phenotype upon growth in a substrate-free medium is in agreement with previous studies which indicated that the cytoplasmic elements do not increase in the absence of galactose

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† Standard Brands Post-doctoral Fellow in Yeast Genetics.

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AN ALGEBRA OF UNBOUNDED OPERATORS

BY J. M. G. FELL AND J. L. KELLEY*

DEPARTMENT OF MATHEMATICS, UNIVERSITY OF CALIFORNIA, AND
DEPARTMENT OF MATHEMATICS, TULANE UNIVERSITY

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This paper is a direct extension of the investigation by M. H. Stone[1] of commutative self-adjoint operator algebras by means of function space techniques. (See also later work by Yosida.[2]) If $\mathfrak{A}$ is such an operator algebra, which is strongly closed, $\mathfrak{A}$ is isomorphic to all continuous complex functions on a certain Hausdorff space X, and to each f of a certain class of unbounded complex functions on X we assign an unbounded normal operator $\bar{T}_f$. The properties of $\bar{T}$ are then studied in detail. Although the proof is omitted for lack of space, each normal operator A is of the form $\bar{T}_f$, where $\mathfrak{A}$ is the algebra generated by $(1 + AA^*)^{-1}A$ and $(1 + AA^*)^{-1}$.

Besides the study of $\bar{T}$, a generalization of a theorem of M. H. Stone[3] on extremally disconnected spaces is proved, and the spectral theorem for unbounded normal operators is a simple consequence of the investigation. The fundamental tool, Lemma 1.1 on monotone convergence, is essentially due to Bochner and Ky Fan.[4]

1. Functional Representation.—Throughout H will be a fixed Hilbert space and $\mathfrak{A}$ will be a commutative algebra of bounded operators on H

which contains the identity 1, is self-adjoint (if $A \in \mathfrak{A}$ then the adjoint $A^* \in \mathfrak{A}$) and is strongly closed (i.e., if $\{A_n\}_n$ is a directed set in $\mathfrak{A}$ and A is a bounded operator such that $\|A_n x - Ax\| \to_n 0$ for each $x \in H$ then $A \in \mathfrak{A}$). Convergence will always be Moore-Smith convergence unless specifically noted otherwise. We write $A \geqq 0$ to mean A is a non-negative operator.

1.1. THEOREM. *If $\{A_n\}_n$ is a monotone increasing directed set of Hermitian operators (i.e., if $m > n$ then $A_m \geqq A_n$) and if for some real k, $k \geqq \|A_n\|$ for all n, then $\{A_n\}_n$ converges strongly to its least upper bound B. Consequently any bounded set of Hermitian members of $\mathfrak{A}$ has a least upper bound which belongs to $\mathfrak{A}$.*

Proof: If A is any bounded non-negative operator then (Ax, y) is an inner product so that, by the Schwartz inequality, $|(Ax, y)|^2 \leqq (Ax, x)(Ay, y)$. Consequently, $\sup\{|(Ax, y)|^2 : \|y\| \leqq 1\} \leqq (Ax, x)\|A\|$, and since $\|Ax\|^2 = |(Ax, Ax/\|Ax\|)|^2 \leqq \sup\{|(Ax, y)|^2 : \|y\| \leqq 1\}$, it is true that $\|Ax\|^2 \leqq (Ax, x)\|A\|$. Considering now the increasing $\{A_n\}_n$, it is clear from the polarization identity that $(A_n x, y)$ converges, for each x, y, and since the limit is a bounded bilinear function of x and y, there is a bounded operator B such that $(A_n x, y) \to_n (Bx, y)$ for all x and y. Since $B - A_n \geqq 0$, from above, $\|(B - A_n)x\|^2 \leqq ((B - A_n)x, x)\|B - A_n\|$ and this converges to zero. The rest of the theorem is simple in view of the functional representation of $\mathfrak{A}$.

If X is the set of multiplicative linear functionals on $\mathfrak{A}$ with the weak * topology then the map F, defined by $F(A)(p) = p(A)$ for $p \in X$, $A \in \mathfrak{A}$, carries $\mathfrak{A}$ isomorphically[6] onto the algebra $\mathfrak{C}$ of complex valued continuous functions on X in such a way that $F(A^*)$ is the conjugate function $F(A)^\sim$. Non-negative operators correspond to non-negative functions. The inverse map to F will always be denoted T, so that for $f \in \mathfrak{C}$, T_f is that operator such that $F(T_f) = f$. Observe that E is a projection if and only if $F(E)$ is the characteristic function of an open and closed set.

If U is any open set in X then the set of all non-negative continuous functions f which are zero outside U and are everywhere $\leqq 1$, has, in view of the preceding theorem, a least upper bound g in $\mathfrak{C}$. It is easy to see that $g = 0$ on $X \setminus U$, and 1 on U and hence 1 on the closure $\bar{U}$ of U. Since g is continuous, $\bar{U}$ is both open and closed. A space such that the closure of every open set is open is *extremally disconnected.*

1.2 X is extremally disconnected, and finite linear combinations of the characteristic functions of open and closed sets are dense in $\mathfrak{C}$.

2. Extremally Disconnected Spaces.—If S is a Borel subset of a topological space there is an open set U such that the symmetric difference $(S \setminus \bar{U}) \cup (U \setminus S)$ is of the first category. If the space is extremally disconnected the set U may be taken open and closed. If the space is com-

pact, so that each open set is of second category, then the corresponding open-closed set U is unique.

For each Borel subset S of X, S' will denote the unique open and closed set such that $[(S \setminus S') \cup (S' \setminus S)] \in$ Cat I.

Given a Borel set S, on considering the open sets S' and $X \setminus S'$, it is clear that $p \in S'$ if and only if for some neighborhood U of p the set $U \setminus S$ is of Cat I. Using this criterion it is easy to see:

2.1. If R and S are Borel sets, $(R \cup S)' = R' \cup S'$, $(R \cap S)' = R' \cap S'$, $R'' = R'$ and $(X \setminus R)' = X \setminus R'$.

M. H. Stone has proved the following theorem for the case Y equals a closed interval of real numbers. A Borel function is a function such that the inverse of a Borel set is a Borel set.

2.2 THEOREM. *If f is a Borel function on X to the compact metric space Y then there is a continuous function g on X to Y such that $f = g$ save on a set of the first category.*

Proof: A subdivision $\mathfrak{F}$ of Y is a finite disjoint family of Borel sets whose union is Y, and *mesh* $\mathfrak{F}$ is the maximum diameter of a member of $\mathfrak{F}$. Choose a sequence $\{\mathfrak{F}_n\}_n$ of subdivisions, each a refinement of its predecessor (each member of $\mathfrak{F}_{n+1}$ is a subset of a member of $\mathfrak{F}_n$) with *mesh* $\mathfrak{F}_n < 1/n$. For each $\mathfrak{F}_n$ choose a function g_n as follows: for each $S \in \mathfrak{F}_n$, g_n is constant on $f^{-1}(S)'$, and on this set takes a value belonging to S. Then g_n is continuous and $|g_n(p) - f(p)| \leqq$ mesh $\mathfrak{F}_n < 1/n$ save on a set of first category. The sequence g_n is a Cauchy sequence since $|g_n(p) - g_{n+m}(p)| \leqq$ mesh $\mathfrak{F}_n$, and converges to a continuous function g, and $|g(p) - g_n(p)| \leqq$ mesh $\mathfrak{F}_n$. The set of all p where $|g(p) - f(p)| > 2/n$ is consequently of the first category,† and the theorem follows.

Let $\bar{\mathfrak{C}}$ be the set of all continuous functions on X to the complex sphere which are ∞ only on a non-dense set. $\bar{\mathfrak{C}}$ is an algebra, if we agree that fg and $f + g$ are those continuous functions which agree with the product and sum save on a set of Cat I. Clearly $\bar{\mathfrak{C}}$ is isomorphic (algebraically) to the algebra of all complex Borel functions modulo the ideal of functions vanishing outside a set of the first category.

3. Extension of T.—For $f \in \bar{\mathfrak{C}}$ let $\mathfrak{F}$ be the set of all characteristic functions of open and closed subsets which are contained in $X \setminus f^{-1}(\infty)$. The set $\mathfrak{F}$ is directed by $\geqq$, and, using 1.1, T_e converges strongly, for $e \in \mathfrak{F}$, to the identity. For $e \in \mathfrak{F}$, $ef \in \mathfrak{C}$.

Let $\bar{T}_f x$ be $\lim \{T_{ef}x : e \in \mathfrak{F}\}$, *whenever this limit exists.* Observe that if e and d belong to $\mathfrak{F}$ and $e \geqq d$ then $\|T_{ef}x\|^2 = (T_{e|f|^2}x, x)$ and $(T_{(e-d)|f|^2}x, x) = \|T_{ef}x\|^2 - \|T_{df}x\|^2$, so that, applying the Cauchy criterion for convergence, either $\|T_{ef}x\|$ converges for $e \in \mathfrak{F}$ to ∞, or it converges to a finite number, in which case $T_{ef}x$ converges to $\bar{T}_f x$.

If $d \in \mathfrak{F}$ then $T_d x$ belongs to the domain of $\bar{T}_f$, for $\|T_{ef}T_d x\|$ is bounded by $\|T_{fd}x\|$. It follows that the domain of $\bar{T}_f$ is dense in H. Moreover,

$T_f T_d x = \lim\{T_{ef} T_d x : e \in \mathfrak{F}\} = \lim\{T_e T_{fd} x : e \in \mathfrak{F}\} = T_{fd} x$, for all $x \in H$. It is also straightforward to see that $T_d \bar{T}_f x = T_{df} x$ for all x in the domain of $\bar{T}_f$.

3.1. MONOTONE CONVERGENCE THEOREM. *If $\{g_n\}_n$ is a directed set of members of $\mathfrak{C}$ which approach $f \in \mathfrak{C}$ monotonically, in the sense that $|f - g_n|$ is monotone decreasing with 0 as lower bound in $\mathfrak{C}$, then $T_{g_n} x$ converges to $\bar{T}_f x$ for each $x \in$ domain $\bar{T}_f$, and $\|T_{g_n} x\|$ converges to ∞ for x not an element of domain $\bar{T}_f$.*

Proof: First, for each $e \in \mathfrak{F}$, $\|(T_{ef} - T_{eg_n})x\|^2 = (T_{e(f-g_n)(f-g_n)\sim} x, x)$ converges to zero with n, because $e|f - g_n|^2$ is a monotonic decreasing directed set and the corresponding operators converge strongly to zero by 1.1. Next, suppose $x \in$ *domain* $\bar{T}_f$, and that g and h are members of $\mathfrak{C}$ such that $|f - g|^2 \geqq |f - h|^2$. Then $\|\bar{T}_f x - T_g x\|^2 = \lim\{\|T_{ef} x - T_{eg} x\|^2 : e \in \mathfrak{F}\} \geqq \lim\{(T_{e(f-h)(f-h)\sim} x, x) : e \in \mathfrak{F}\} = \|\bar{T}_f x - T_h x\|^2$. It follows that if $m \geqq n$ and $d \geqq e$ then $\|\bar{T}_f x - T_{dg_m} x\| \geqq \|\bar{T}_f x - T_{eg_n} x\|$. Now, for $x \in$ *domain* $\bar{T}_f$, choose $e \in \mathfrak{F}$ so $T_{ef} x$ is near $\bar{T}_f x$, then n so that if $m \geqq n$ then $T_{eg_m} x$ is near $T_{ef} x$ and hence near $\bar{T}_f x$. As e converges over $\mathfrak{F}$ to 1, the distance $\|\bar{T}_f x - T_{eg_m} x\|$ decreases, so that $\|\bar{T}_f x - T_{g_m} x\|$ is small for $m \geqq n$. If x is not an element of *domain* $\bar{T}_f$, choose $e \in \mathfrak{F}$ so $T_{ef} x$ is large whence for some n, if $m \geqq n$, $\|T_{eg_m} x\|$ is large, and since this norm is monotonic increasing in e, $\|T_{g_m} x\|$ is large.

A particular consequence of the theorem is that, in computing $\bar{T}_f x$, $\mathfrak{F}$ may be replaced by any subset $\mathcal{E}$ of $\mathfrak{F}$ which is directed by $\geqq$ and such that T_e, for $e \in \mathcal{E}$, converges strongly to the identity.

3.2. THEOREM. *$\bar{T}_f$ is normal, and its adjoint operator is $\bar{T}_{f\sim}$.*

Proof: Since $x \in$ *domain* $\bar{T}_f$ if and only if $\|T_{ef} x\|$ is uniformly bounded for $e \in \mathfrak{F}$, and since $\|T_{ef\sim} x\| = \|T_{ef} x\|$, it is clear that *domain* $\bar{T}_{f\sim}$ = *domain* $\bar{T}_f$. Also $\|\bar{T}_f x\| = \lim\{\|T_{ef} x\| : e \in \mathfrak{F}\} = \lim\{\|T_{ef\sim} x\| : e \in \mathfrak{F}\} = \|\bar{T}_{f\sim} x\|$, so it remains to prove that $\bar{T}_{f\sim}$ is the adjoint of $\bar{T}_f$. Suppose $(\bar{T}_f x, y) = (x, y^*)$ for each $x \in$ *domain* $\bar{T}_f$. Then $(\bar{T}_f x, y) = \lim\{(T_{ef} x, y) : e \in \mathfrak{F}\} = \lim\{(x, T_{ef\sim} y) : e \in \mathfrak{F}\}$, and it will follow that $y^* = \bar{T}_{f\sim} y$ if it can be shown that $\|T_{ef\sim} y\|$ is bounded for $e \in \mathfrak{F}$. Let $x = T_{df\sim} y$ where $d \in \mathfrak{F}$. Then $(T_{df\sim} y, y^*) = \lim\{(T_{ef} T_{df\sim} y, y) : e \in \mathfrak{F}\} = \lim\{(T_e T_{df f\sim} y, y) : e \in \mathfrak{F}\} = \|T_{df\sim} y\|^2$. Dividing by $\|T_{df\sim} y\|$, it is clear that $\|T_{df\sim} y\| \leqq \|y^*\|$, and the theorem is established.

3.3. THEOREM. *If $\{x_n\}_n$ is a sequence in domain $\bar{T}_f$, if $\|x_n - x\| \to_n 0$ and $\|\bar{T}_f x_n\|$ is bounded, then $\bar{T}_f x_n$ converges weakly to $\bar{T}_f x$. If x is not an element of domain $\bar{T}_f$ then $\|\bar{T}_f x_n\| \to_n \infty$.*

Proof: Suppose $\|\bar{T}_f x_n\| \leqq k$ for each n. Since for $e \in \mathfrak{F}$, $\|T_{fe} x_n\| \leqq \|\bar{T}_f x_n\| \leqq k$ then, letting $n \to \infty$, $\|T_{fe} x\| \leqq k$ for each $e \in \mathfrak{F}$ and hence $x \in$ *domain* $\bar{T}_f$. Since $\bar{T}_f x_n$ is a bounded sequence, weak convergence to $\bar{T}_f x$ will be proved if $(\bar{T}_f x_n, y) \to_n (\bar{T}_f x, y)$ for y belonging to a dense subset of H. Let $y = T_e z$, for $e \in \mathfrak{F}$. Then $(\bar{T}_f x_n, T_e z) = (T_e \bar{T}_f x_n, T_e z) =$

$(T_{ef}x_n, T_ez) \to_n (T_{ef}x, T_ez) = (T_e\bar{T}_fx, T_ez) = (\bar{T}_fx, T_ez)$. To prove the second statement, if $e \in \mathfrak{F}$ so that $\|T_{ef}x\|$ is large, then for n large, $\|T_{ef}x_n\|$ is large, and $\|T_{ef}x_n\| \leqq \|\bar{T}_fx_n\|$.

This theorem strengthens the result that the graph of $\bar{T}_f$ is closed.

We now show the closure of the graph of $\bar{T}_f\bar{T}_g$ is the graph of $\bar{T}_{gf}$. Let $\mathcal{E}$ be the set of characteristic functions of all open and closed sets which are disjoint from $f^{-1}(\infty) \cup g^{-1}(\infty)$. Then $\bar{T}_fx$, $\bar{T}_gx$ and $\bar{T}_{fg}x$ can each be computed as $\lim \{T_{ef}x : e \in \mathcal{E}\}$, etc. If $\bar{T}_gx \in$ domain $\bar{T}_f$ then for $e \in \mathcal{E}$, $\bar{T}_f\bar{T}_gx = \lim \{\bar{T}_fT_{eg}x : e \in \mathcal{E}\} = \lim \{\bar{T}_fT_eT_{eg}x : e \in \mathcal{E}\} = \lim \{T_{efg}x : e \in \mathcal{E}\}$, and since this limit exists, it is $\bar{T}_{fg}x$. The graph of $\bar{T}_f\bar{T}_g$ is then a subset of the graph of $\bar{T}_{fg}$. For $x \in$ domain $\bar{T}_{fg}$, choose $e \in \mathcal{E}$ so that x is near T_ex and $T_{efg}x$ is near $\bar{T}_{fg}x$. Then $\bar{T}_gT_ex = T_eT_{eg}x$ belongs to domain $\bar{T}_f$, and since $\bar{T}_f\bar{T}_gT_ex = T_{efg}x$ the pair $\langle T_ex, \bar{T}_f\bar{T}_gT_ex\rangle$ is near the pair $\langle x, \bar{T}_{fg}x\rangle$. The same sort of argument shows that the graph of $\bar{T}_{f+g}$ is the closure of the graph of $\bar{T}_f + \bar{T}_g$ and the following theorem is established.

3.4. THEOREM. *The algebra $\bar{\mathcal{C}}$ is isomorphic to an algebra $\bar{\mathfrak{A}}$ under $\bar{T}$, in the sense that the graphs of $\bar{T}_{fg}$ and $\bar{T}_{f+g}$ are, respectively, the closures of the graphs of $\bar{T}_f\bar{T}_g$ and $\bar{T}_f + \bar{T}_g$.*

If $f \in \bar{\mathcal{C}}$ and the set of zeros of f contains an open and closed set then $ef = 0$ for the characteristic function e of this set. Each x belonging to the range of T_e maps under $\bar{T}_f$ into zero, so that $\bar{T}_f$ can have no inverse. If the zeros of f are non-dense we will now show that $\bar{T}_{1/f}\bar{T}_fx = x$ for each $x \in$ *domain* $\bar{T}_f$. If e is the characteristic function of an open and closed set disjoint from $(1/f)^{-1}(\infty)$, $T_{e(1/f)}\bar{T}_fx = T_{e(1/f)}T_e\bar{T}_fx = T_ex$. Consequently, the norm of this vector is bounded, for such an e, by $\|x\|$ and $\bar{T}_fx \in$ *domain* $\bar{T}_{1/f}$, and the preceding theorem completes the proof.

3.5. *$\bar{T}_f$ has an inverse if and only if the zeros of f are non-dense, and in this case $(\bar{T}_f)^{-1} = \bar{T}_{1/f}$.*

4. The Spectral Resolution for $\mathfrak{A}$.—The following form of the spectral theorem is suggested by the measure theoretic form used by Halmos.[5] *For each Borel set U in X let $\mu(U) = T_e$, where e is the characteristic function of the open and closed set U' such that the symmetric difference $(U' \setminus U) \cup (U \setminus U')$ is of first category.* The projection valued set function μ is the *spectral measure* for $\mathfrak{A}$ (or for $\bar{\mathfrak{A}}$). If $\mathcal{U}$ is a family of Borel subsets of X we agree that $\Sigma\{\mu(U) : U \in \mathcal{U}\}$ is to be the limit in the strong operator topology of the sums over finite subfamilies of $\mathcal{U}$, the limit being taken in the direction of increasing subfamilies.

4.1. *μ is additive over countable disjoint families of Borel sets, and over arbitrary disjoint families of open sets.*

Proof: If $\mathcal{U}$ is a disjoint family of Borel sets and $\mathcal{U}'$ is the family of corresponding open and closed sets then the finite sums defining $\Sigma\{\mu(U) : U \in \mathcal{U}\}$ are a monotone increasing directed set of projections bounded by the identity, and consequently converge in the strong operator

topology to their least upper bound. This least upper bound is the correspondent under T of the characteristic function e of the closure of $\cup\{U: U \in \mathfrak{U}'\}$. If the members of $\mathfrak{U}$ are open, or if $\mathfrak{U}$ is countable, T_e is $\mu(\cup\{U: U \in \mathfrak{U}'\})$.

For a Borel set U in X let $\mathfrak{U}$ be the family of all open sets which contain U. Then $\mathfrak{U}$ is directed by $\subset$, and we will show that $\mu(U)$ is the limit in the strong operator topology of $\mu(V)$ for $V \in \mathfrak{U}$. First, in case U is non-dense, $\lim\{\mu(V): V \in \mathfrak{U}\}$ is 0, because of 1.1, and for each $x \in H$, $\|\mu(V)x\| \to_V 0$. Next, if U is the union of a sequence of non-dense sets, we may, by the familiar "$\epsilon/2^n$" argument, again see that $\|\mu(V)x\| \to_V 0$ for each $x \in H$. The result, for arbitrary Borel set U, is now immediate. By complementation it follows that $\mu(U)$, for each Borel set U, is the strong limit of $\mu(C)$ for compact subsets C of U directed by $\supset$. A set function ν, on the Borel sets of X to a topological space L, such that $\nu(U)$ is the limit of $\nu(V)$ for V open and containing U is *regular* relative to the topology of L. The following theorem is due to Halmos.[5]

4.2. μ is regular relative to the strong topology for $\mathfrak{A}$. If G is a function on $\mathfrak{A}$ to a topological space which is continuous relative to the strong topology then $G\mu$ is regular.

For $f \in \bar{\mathfrak{C}}$ and ν a finitely additive function on the Borel sets of X to a linear topological space we define $\int f d\nu$ as follows. (The integral depends on the topology assigned the linear topological space.) If f is the characteristic function of a Borel set U then $\int f d\nu = \nu(U)$. A *simple* function is a finite linear combination of characteristic functions of Borel sets, and its integral is defined accordingly. For $f \in \mathfrak{C}$, $f \geqq 0$, let $\mathfrak{G}$ be the set of all simple functions g such that $o \leqq g \leqq f$. Then $\mathfrak{G}$ is directed by $\geqq$, and $\int f d\nu$ is the limit of $\int g d\nu$ for $g \in \mathfrak{G}$. The integral of an arbitrary real function belonging to $\bar{\mathfrak{C}}$ is the difference of the integrals of the positive and negative parts, and the integral of a complex function is (*integral of real part*) $+ i$ (*integral of imaginary part*). It is straightforward to verify that $T_f = \int f d\mu$ for $f \in \mathfrak{C}$. (The limit defining the integral is taken in the norm topology for $\mathfrak{A}$.)

For $x \in H$ let μ^x be the set function such that for each Borel set U, $\mu^x(U) = \mu(U)x$. Then μ^x has values in H. By using the theorem 3.1 on monotone convergence, and noticing that if $x \in$ *domain* T_f then x belongs to the domain of the operators corresponding to each of the four parts of f, one obtains:

4.3. SPECTRAL THEOREM. *For $f \in \mathfrak{C}$, $T_f = \int f d\mu$. For $f \in \bar{\mathfrak{C}}$, $\bar{T}_f x = \int f d\mu^x$. ($x \in$ domain $\bar{T}_f$ if and only if the right-hand integral exists.)*

For $y \in H$, let $\mu^{x,y}(U) = (\mu^x(U), y) = (\mu(U)x, y)$. Then $\mu^{x,y}$ is a finite, complex valued "measure," and $(\bar{T}_f x, y) = \int f d\mu^{x,y}$. The definition of integral coincides, for $\mu^{x,y}$, with the conventional one. However, 4.3 is

stronger than the latter result—the sums approximating $\int f d\mu^x$ converge to $T_f x$ in the norm topology of H, as well as in the weak.

For $f \in \mathcal{C}$ we define a spectral measure on the Borel subsets of the complex plane by $\nu(U) = \mu(f^{-1}(U))$. It is easy to check that for U open $\nu(U)$ is the strong limit of the measures of compact subsets of U, so that ν is regular. Moreover, $\int f(p) d\mu p = \int Z d\nu Z$, for one may verify that the approximating sums are identical. Hence, $T_f = \int Z d\nu Z$ for $f \in \mathcal{C}$, and $T_f x = \int Z d\nu^x Z$ for $f \in \mathcal{C}$, where $\nu^x(U) = \nu(U)(x)$.

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† It is precisely this point at which the requirement that Y be metric, seems essential. If Y is not metric, but compact Hausdorff, a modification of the argument given here shows that $f = g$ save on a set S with the property: if ρ is any pseudometric on X, then S is of first category with respect to the topology given by ρ.

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COMMUTATIVE OPERATOR ALGEBRAS

BY J. L. KELLEY*

DEPARTMENT OF MATHEMATICS, TULANE UNIVERSITY, NEW ORLEANS, LA.

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This is an investigation of the structure of a commutative strongly closed, self adjoint algebra $\mathfrak{A}$, with $1 \in \mathfrak{A}$, of operators on a Hilbert space H. For brevity, the paper is written as a continuation of the preceding note[1] assuming its notation and results. The principal theorem is: There is a unique multiplicity function ϕ on the spectrum X of $\mathfrak{A}$ to the cardinal numbers, ϕ being continuous relative to the order topology for the cardinals; from X and ϕ the space H and the algebra $\mathfrak{A}$ may be reconstructed, to a unitary equivalence. (The intuitive motivation: If $x \in H$ is, for each $A \in \mathfrak{A}$, a characteristic vector with characteristic value $\lambda(A)$, then λ is a homomorphism of $\mathfrak{A}$ into the complex numbers; i.e., $\lambda \in X$. Thus X can be interpreted as the space of characteristic values, to each of which we

assign a multiplicity.) This theorem may be compared with two treatments of multiplicity theory which have appeared since the body of this paper was written. Segal[2] exhibits, as a complete unitary invariant, a function B which to each cardinal n assigns a measure algebra $B(n)$; explicitly, $B(n)$ is the Boolean algebra of open and closed subsets of the closure of the interior of $\phi^{-1}(n)$, and it is equally easy to describe X and ϕ in terms of the function B. However, we obtain more explicit results (2.3, 2.4) on the structure of $\mathfrak{A}$ than were previously known, we derive weak from strong closure of $\mathfrak{A}$, and the reconstruction of $\mathfrak{A}$ and H from the invariants is in part novel (although 5.3 should be compared with [Ref. 2, p. 63 Remark 9.2]). The comparison with Halmos[3] is more difficult since he does not explicitly consider an algebra of operators. However, from his work it follows that the Boolean algebra of all projections E belonging to $\mathfrak{A}$, together with a multiplicity $m(E)$, is a complete unitary invariant. The relation to X, ϕ is the obvious one: $m(E)$ is the minimum of ϕ on the open and closed subset of X corresponding to E. Given a single normal operator A the theory we give suggests the obviously complete invariant (X, ϕ, f) where $T_f = A$. However, a simplification can be made (Remark 2.5) in this case, which will give Halmos' form of the Wecken-Plesner-Rohlin theorem.

I am indebted to J. M. G. Fell for several conversations, and for the proof of 3.2. In the following the measure—theoretic notation—is that of Halmos.[4]

1. Structure on $\mathfrak{A}x$.—Recall that μ is the spectral measure for $\mathfrak{A}$. For each $x \in H$ and each Borel subset U of X let $\mu_x(U) = (\mu(U)x, x)$. If e is the characteristic function of the open and closed subset of X whose symmetric difference with U is of Cat I then $(\mu_x(U) = (T_e x, x)$. From the properties of μ it follows that μ_x is a regular measure, vanishing on the sets of the first category, and is additive over arbitrary disjoint families of open sets. If V is the complement of the closure of the union of a maximal disjoint family of open-closed sets of measure zero then: each non-void open-closed subset of V has non-zero measure and each subset of $X \setminus V$ has measure zero. The unique open and closed set with these two properties is the *carrier* of μ_x. If f is the characteristic function of an open and closed set then $\int f d\mu_x = (T_f x, x)$, and since linear combinations of such f's are dense in $\mathfrak{C}$, equality holds for each $f \in \mathfrak{C}$.

For f and g in $\mathfrak{C}$, $(T_f x, T_g x) = (T_{f\bar{g}} x, x) = \int f\bar{g}\, d\mu_x$. That is, for fixed x, T maps $\mathfrak{C}$ into H in such a way that the $L_2(\mu_x)$ inner product is preserved. *Let $\mathfrak{I}_x$ be the unitary extension of this map which carries $L_2(\mu_x)$ onto $\overline{\mathfrak{A}x}$* (the closure of $\mathfrak{A}x$).

1.1. For each $f \in \bar{\mathfrak{C}}$ and each $x \in H$, $\bar{T}_f x = \mathfrak{I}_x f$ whenever either is defined.

Proof: Let $\{e_n\}_n$ be a monotone increasing sequence of characteristic functions of open and closed sets, each disjoint from $f^{-1}(\infty)$, such that

T_{e_n} converges strongly to 1. (Such can be obtained by taking inverses of neighborhoods of ∞.) Then $f \in L_2(\mu_x)$ if and only if $\lim \int |fe_n|^2 d\mu_x < \infty$ (monotone convergence of integrable functions), and this is also necessary and sufficient that $x \in$ *domain* $\bar{T}_f$. If $x \in$ *domain* $\bar{T}_f$, then $e_n f \to_n f$ in $L_2(\mu_x)$ and $\mathfrak{I}_x e_n f = T e_n f x \to_n \bar{T}_f x$ in H, whence the theorem.

In particular, *domain* $\bar{T}_f$ is the set of all x such that $f \in L_2(\mu_x)$, and if $y \in \bar{\mathfrak{A}}_x$ then, for some $f \in L_2(\mu_x)$, $y = \bar{T}_f x$.

For each $f \in \bar{\mathcal{C}}$ let $M_f g = fg$. (M_f is *multiplication by* f.) For f and g in $\mathcal{C}$, $T_f \mathfrak{I}_x g = T_f T_\varphi x = \mathfrak{I}_x M_f g$. We now show that $\bar{T}_f$ on $\mathfrak{A}x$ is unitarily equivalent to M_f on $L_2(\mu_x)$.

1.2 THEOREM. *For* $y \in \bar{\mathfrak{A}}_x$, $\bar{T}_f y = \mathfrak{I}_x M_f \mathfrak{I}_x^{-1} y$.

Proof: Because both $\bar{T}_f$ and M_f satisfy the continuity theorem (Ref. 1, 3.3) it is sufficient to verify equality for y in a set dense in the domain of each operator. One such set consists of vectors $T_\varphi x$, where $g \in \mathcal{C}$ and g vanishes on a neighborhood of $f^{-1}(\infty)$, and in this case $\mathfrak{I}_x M_f \mathfrak{I}_x^{-1} T_\varphi x = \mathfrak{I}_x M_f g = T_{fg} x = \bar{T}_f T_\varphi x$.

2. Structure of $\mathfrak{A}$.—We agree that E *carries* x, or is the carrier of x, if $x \in H$ and E is the infimum of all those projections in $\mathfrak{A}$ whose range contains x. Clearly, if e is the characteristic function of the carrier of μ_x, T_e carries x. For $f \in \bar{\mathfrak{A}}$, the carrier of the measure corresponding to $\bar{T}_f x$ is the intersection of the carrier of μ_x with the closure of the set of all p such that $f(p) \neq 0$. This establishes the first half of the following, and the second is straightforward.

2.1. If E *carries* x *then for each projection* F *in* $\mathfrak{A}$, EF *carries* Fx. *If* $\{x_n\}_n$ *is a sequence,* $\|x_n\| \leqq 1$, E_n *carries* x_n *and* $E_n E_m = 0$ *for* $n \neq m$ *then* $\Sigma_n E_n$ *carries* $\Sigma_n x_n/n$.

2.2 An open and closed set U *is the carrier of* μ_x *for some* $x \in H$ *if and only if* U *satisfies the countable chain condition: i.e., each disjoint family of open subsets of* U *is countable.*

Proof: It is clear that the carrier of μ_x satisfies the condition. Suppose U open and closed. Then U contains the carrier of some μ_x (let $x \in$ range T_e where e is the characteristic function of U), and consequently, the union of any maximal disjoint family of carriers, each contained in U, is dense in U. If such a family is countable, by 2.1 U is the carrier of some μ_x.

For convenience, a set is defined to be a *restricted Borel set* if it is a Borel subset of some open and closed set which satisfies the countable chain condition.

If $y = \bar{T}_f x$ then for $g \in \mathfrak{A}$, $\int g d\mu_y = \int |f|^2 g d\mu_x$, so that μ_y is "μ_x with weighting factor $|f|^2$" and we write $\mu_y = |f|^2 \cdot \mu_x$. If η is a finite Borel measure which is absolutely continuous with respect to μ_x, then by the Radon-Nikodym theorem, for some non-negative $f \in \bar{\mathcal{C}}$, $\eta = f \cdot \mu_x$ and η is the measure corresponding to $\bar{T}_\varphi x$ where $g = (f)^{1/2}$. If $\eta \leqq \mu_x$ then for some bounded f, $\eta = f \cdot \mu_x$. Finally, recall that for a Borel set U, $\mu_x(U) = 0$

if and only if the intersection of U with the carrier of μ_x is of category I. Parts (a) and (b) of the following are then clear.

2.3. THEOREM. *(a) If η is a Borel measure absolutely continuous w.r.t. μ_x then for some $A \in \mathfrak{A}$, $\eta = \mu_{Ax}$. If $\eta \leqq \mu_x$ then for some $A \in \mathfrak{A}$, $\eta = \mu_{Ax}$.*

(b) If carrier $\mu_y \subset$ carrier μ_x then for some $z \in \mathfrak{A}x$, $\mu_z = \mu_y$.

(c) If carrier $\mu_y =$ carrier μ_x and $y \in \mathfrak{A}x$ then $\mathfrak{A}x = \mathfrak{A}y$.

(d) If a finite Borel measure η vanishes on sets of Cat I, and if for some restricted Borel set U, $\eta(X) = \eta(U)$ then $\eta = \mu_x$ for some $x \in H$.

Proof: To prove (c) note that $y = T_f x$ where we may suppose that $f = 1$ outside *carrier* μ_x, and from the conditions on carriers it follows that the set of zeros of f is of Cat I, so by Ref. 1, 3.5, $x = T_{1/f} y$. To prove (d) observe that U may be taken open and closed, and since U satisfies the countable chain condition, some open and closed subset of U is the carrier of η and is also the carrier of some μ_x. Then η is absolutely continuous w.r.t. μ_x, so that (a) applies.

From (d) it is easy to prove the interesting (but for our purposes, inessential) fact that a finite Borel measure is μ_x for some x if and only if it is completely additive over the Boolean algebra of open and closed subsets of X. We shall call measures of the form μ_x *characteristic* measures; they are determined by X and its topology. By means of the characteristic measures we define a σ-finite measure ν on the σ-ring of all restricted Borel sets. Choose a maximal set M of characteristic measures having disjoint carriers, and set $\nu(U) = \Sigma\{\eta(U) : \eta \in M\}$ for each restricted Borel set U. Because the union of the carriers of members of M is dense in X, ν is definitely positive on open sets. Moreover, a Borel set V is of Cat I if and only if $\nu(U \cap V) = 0$ for each restricted Borel set U. Defining *ess sup* $f =$ *inf* k: such that $\nu\{p : |f(p)| \geqq k\} \cap U) = 0$ for each restricted Borel U, it follows that $\mathfrak{C}$ is isomorphic to the space of essentially bounded Borel functions. It may be verified directly that $\mathfrak{C}$ is the adjoint of $L_1(\nu)$ and finally, using 2.3(d), that a measure η is a characteristic measure if and only if for some non-negative $f \in L_1(\nu)$, $\eta = f \cdot \nu$ on restricted Borel sets. (Although ν is not totally σ-finite one easily establishes the Radon-Nikodym theorem for measures absolutely continuous w.r.t. ν.) The *weak* topology* for $\mathfrak{C}$ is its weak* topology as the adjoint of $L_1(\nu)$. Because of the form of the members of $L_1(\nu)$, a directed set $\{f_n\}_n$ converges weak* to f if and only if $(T_{f_n} x, x) \to_n (T_f x, x)$ for each $x \in H$ so that

2.4. THEOREM. *The algebra $\mathfrak{A}$ is isomorphic under T to the algebra of ν-essentially bounded functions on X. The weak operator topology for $\mathfrak{A}$ corresponds precisely to the weak* topology for $\mathfrak{C}$. For $f_n \in \mathfrak{C}$, T_{f_n} converges strongly to T_f if and only if $|f_n - f|^2$ converges weak* to zero.*

It is also clear that pointwise convergence of a bounded *sequence* of members of $\mathfrak{C}$ implies strong convergence of the corresponding operators, by Lebesgués' bounded convergence theorem. Finally, note that the

unit sphere of $\mathcal{C}$ is weak* compact, whence, if $\mathfrak{A}'$ is the weak closure of $\mathfrak{A}$ and $\mathcal{C}'$ is the corresponding function algebra, the intersection of $\mathcal{C}$ with the unit sphere of $\mathcal{C}'$ is weak* compact (weak* convergence in $\mathcal{C}'$ also corresponds to weak operator convergence) so that $\mathcal{C}$ is weak* closed in $\mathcal{C}'$ by a theorem of Krein and Smulian[5] and (a theorem of von Neumann[6]):

2.5. THEOREM. *$\mathfrak{A}$ is weakly closed in the algebra of all bounded operators on H.*

2.6. Remark. Let $\mathfrak{M}$ be the set of all finite linear combinations of characteristic measures on X. It is not hard to see that X is determined, to a homeomorphism, by the ordered Banach space $\mathfrak{M}$. (Indeed, as a part of the general theory of L-spaces (for example, [F]), each of $\mathcal{C}$, $L_1(\nu)$, $\mathfrak{M}$, X and the Boolean algebra of open and closed subsets of X determines, to a natural equivalence, the others.) If now, $\mathfrak{A}$ is the smallest strongly closed, self-adjoint algebra containing a single normal operator $A = T_f$ and the identity operator, then: f carries X onto the spectrum $\sigma(A)$, thereby inducing an isometry of the continuous complex functions on $\sigma(A)$ onto a weak* dense subalgebra of $\mathfrak{A}$, so that the map induced on Borel measures by f is, on $\mathfrak{M}$, an order-preserving isometry. Consequently in this case (and actually in this case only) there is a concrete representation of $\mathfrak{M}$ as a set of signed Borel measures on the complex plane, and this set may be used instead of X as an invariant.

3. $\mathfrak{A}$ Bases and the Multiplicity Function.—Treating $\mathfrak{A}$ as the scalars, H can be decomposed in a direct extension of the usual representation in terms of a complete orthonormal set. Two subsets of H are orthogonal, or $\perp$, if each member of one is $\perp$ to each member of the other. Observe that $x \perp \mathfrak{A}y$, $\mathfrak{A}x \perp \mathfrak{A}y$, $\overline{\mathfrak{A}x} \perp y$, etc., are all equivalent. If $x \perp \mathfrak{A}y$ then $(E(x + y), x + y) = (Ex, x) + (Ey, y)$ so that $\mu_{x+y} = \mu_x + \mu_y$. Consequently, if E carries x and F carries y and $x \perp \mathfrak{A}y$ then $x + y$ is carried by $E + F - EF$. Hence, if $x \in$ *range* F and $x = y + z$ where $y \perp \mathfrak{A}z$ then y and $z \in$ *range* F. We now define an *$\mathfrak{A}$ base for a projection* E belonging to $\mathfrak{A}$ to be a non-void subset J of H such that: (*a*) if $x \in J$ then $0 \neq \|x\| \leqq 1$ and E carries x, (*b*) if x and y are distinct members of J then $x \perp \mathfrak{A}y$ and (*c*) J is maximal with respect to (*a*) and (*b*). It is clear that any projection which is the carrier of some x of H has at least one $\mathfrak{A}$ base. The smallest closed linear subset of H containing $\mathfrak{A}x$ for each $x \in J$ is denoted $\mathfrak{A}\{J\}$.

3.1. If J is an $\mathfrak{A}$ base for E then for some projection F, $0 \neq F \leqq E$, and range $F \subset \mathfrak{A}\{J\}$. For such an F, the set K of vectors Fx for $x \in J$ is an $\mathfrak{A}$ base for F and $\mathfrak{A}\{K\} =$ range F.

Proof: Suppose that $\mathfrak{A}\{J\}$ contains the range of no non-zero projection which is $\leqq E$. Then for each projection F, $0 \neq F \leqq E$, there is $x \in$ *range* F, x not an element of $\mathfrak{A}\{J\}$, and $x = y + z$ where $y \neq 0$, $y \perp \mathfrak{A}\{J\}$ and $z \in \mathfrak{A}\{J\}$. Then since $\mu_x = \mu_y + \mu_z$, y is carried by some projection F_0,

and $0 \neq F_0 \leqq F \leqq E$. Choose a maximal set K of unit vectors y, such that $y \perp \mathfrak{A}\{J\}$, $y \in$ *range* E, and the family $\mathfrak{F}$ of carriers of members of K is disjoint. Then, from above; it follows that $\Sigma\{F: F \in \mathfrak{F}\} = E$. Now $\mathfrak{F}$ is countable, whence $\Sigma\{a_x x: x \in K\}$, where the coefficients a_x are non-zero and $\Sigma\{|a_x|^2: x \in K\} \leqq 1$, is carried by E and is orthogonal to $\mathfrak{A}\{J\}$, which is a contradiction to the maximality of J. The latter statement of the theorem is straightforward.

A projection E is *primitive* if there is an $\mathfrak{A}$ base J for E such that $\mathfrak{A}\{J\} =$ *range* E. If $\mathfrak{A}\{J\} =$ *range* E, and further, $\mu_x = \mu_y$ for all x and y in J then J is a *proper* $\mathfrak{A}$ base for E. Because of 2.3 each primitive projection has a proper $\mathfrak{A}$ base.

3.2. If J and K are proper $\mathfrak{A}$ bases for E then $\mathbf{P}(J) = \mathbf{P}(K)$, *where* $\mathbf{P}$ *is "the cardinal number of."*

Proof: The procedure is a mild variant of the usual proof for the case $\mathfrak{A} =$ scalars. Suppose first that both J and K are infinite. For $x \in J$ let L_x be the set of all y in K such that y is not $\perp \mathfrak{A}x$. The linear spaces $\mathfrak{A}y$, $y \in K$, are mutually orthogonal, so for each $x \in J$, $x \perp \mathfrak{A}y$ save for a countable set of y's; consequently $\mathfrak{A}x \perp y$ save for a countable set and L_x is countable. Since each y of K belongs to at least one L_x we have $\mathbf{P}(K) \leqq \mathbf{P}(J \times (\text{countable set})) = \mathbf{P}(J)$ and by symmetry $\mathbf{P}(J) = \mathbf{P}(K)$. In case J is finite, consisting of $x_1, \ldots x_n$, and if $y_1, \ldots y_m$ are members of K, it will be shown that $m \leqq n$. We may suppose that $\mu_{x_i} = \mu_{y_j}$ for all i, j. If y_i is written as $u + v$, where $u \in \mathfrak{A}_{x_j}$ and $v \perp \mathfrak{A}_{x_j}$ then $\mu_{y_i} = \mu_u + \mu_v$ and, consequently, by 2.3, $u = Bx_j$ for some $B \in \mathfrak{A}$. It follows that there are $A_{ij} \in \mathfrak{A}$ such that $y_i = \Sigma\{A_{ij}x_i: j = 1, \ldots n\}$, and it may be assumed that $EA_{ij} = A_{ij}$. Since $y_i \perp \mathfrak{A}y_k$ for $i \neq k$, then for all $A \in \mathfrak{A}$, $\Sigma\{(AA_{ki}{}^*A_{ij}x_j, x_j): j = 1, \ldots n\}$ is 0 for $k \neq i$, and is (Ay_i, y_i) for $k = i$. Upon considering the functional representation, it becomes clear that $\Sigma_j A_{kj}{}^*A_{ij}$ is 0 for $k \neq i$ and E for $k = i$. Whence each x may be expressed in terms of the first n of the y's and necessarily $m \leqq n$.

Notice that, by 3.1, if E and F are primitive projections then EF is primitive or zero, and if $EF \neq 0$ then the cardinal of a proper $\mathfrak{A}$ base for EF is the same as that of an $\mathfrak{A}$ base for E. Moreover, by 3.1 again, if $\mathfrak{F}$ is a maximal disjoint set of primitive projections then $\Sigma\{F: F \in \mathfrak{F}\}$ is the identity 1. In terms of such an $\mathfrak{F}$ the structure of $\mathfrak{A}$ and H can be described: If, for $F \in \mathfrak{F}$, J_F is a proper $\mathfrak{A}$ base for F, then H is the Hilbert space project of $\mathfrak{A}x$, for $x \in \cup\{J_F: F \in \mathfrak{F}\}$ and on $\mathfrak{A}x$ the behavior of $\mathfrak{A}$ is known by 1.2.

If e is the characteristic function of an open and closed set to which a point of p of X belongs, then $\phi_1(p)$ is defined to be the cardinal of a proper $\mathfrak{A}$ base for T_e, when such a base exists. From above, ϕ_1 is defined unambiguously on a dense open subset of X. If the cardinals (or the cardinals $\leqq \mathbf{P}(H)$) are given the order topology, i.e., the topology with

"open intervals" as a base, then ϕ_1 is continuous. By using the fact that, for each cardinal c, the sets of all p with $\phi_1 < c$ and $\phi_1 > c$, respectively, are disjoint and open, and consequently have disjoint closures, it can be seen directly that ϕ_1 has a continuous extension ϕ to all of X. ϕ is the *multiplicity function for* $\mathfrak{A}$. X and ϕ are unitary invariants in the sense that: A unitary operator on H onto a Hilbert space H' induces an isomorphism of $\mathfrak{A}$ onto an algebra $\mathfrak{A}'$, which induces a homeomorphism h of the spectrum of $\mathfrak{A}'$ on to X' and the multiplicity function for $\mathfrak{A}'$ is the composition ϕh. Finally, notice that for any continuous function ψ on X to the cardinals, the set of p where $\psi(p)$ is a minimum is open; from this it follows that there is a disjoint family of open subsets of X, on each of which ψ is constant, such that the union of the family is dense in X.

4. *Structure of* $\mathfrak{A}$ *on the Range of a Primitive Projection.*—The behavior of $\mathfrak{A}$ on the range of a primitive projection can be described in three ways, via the usual correspondences between $(m^n)^p$, $m^{n\times p}$ and $(m^p)^n$ for sets m, n and p. For notational convenience we suppose throughout this section that the identity 1 is the primitive projection, that J is a proper $\mathfrak{A}$ base for 1 so that $\mathfrak{A}\{J\} = H$, and that $\mu_x = \mu_y = \nu$ for x and y belonging to J. The *counting measure* γ on J is the measure, defined on the σ-ring of countable subsets of J, such that $\gamma(\{x\}) = 1$ for each $x \in J$.

First, it is clear that H is isomorphic to the product of $\bar{\mathfrak{A}}_x$, $x \in J$, and each factor is isomorphic to $L_2(\nu)$. If Θ is the set of all functions θ on J to $L_2(\nu)$ such that $\Sigma\{\|\theta_x\|^2 : x \in J\} = \|\theta\|^2 < \infty$, then Q, where $Q\theta = \Sigma\{\Im_x\theta_x : x \in J\}$ is a unitary map of Θ onto H, and T_f on H corresponds to coordinate wise multiplication by f, i.e., $(Q^{-1}T_fQ\theta)_x = f\theta_x$, because of 1.2.

Second, let $\nu \times \gamma$ be the product measure of ν on X and γ on J. If $k \in L_2(\nu \times \gamma)$, set $k_x(p) = k(p, x)$ for each $p \in X$, $x \in J$. Observe, from the Fubini theorem, that $\int |k|^2 d\nu \times \gamma = \Sigma\{\int |k_x|^2 d\nu : x \in J\}$ so that $L_2(\nu \times \gamma)$ is unitarily equivalent to Θ under the map that sends k into the member of Θ whose value at x is k_x. If $Rk = \Sigma\{\Im_x k_x : x \in J\}$, R is unitary and $(R^{-1}T_fRk)(p, x) = f(p)k(p, x)$.

Third, let Ψ be the set of all Borel functions ψ on X to $L_2(\gamma)$ such that the range of ψ is separable (i.e., ψ^{-1} of each open set is a Borel set, and there is a countable dense subset of the image of X under ψ) and such that $\|\psi\|^2 = \int \|\psi(p)\|^2 d\nu p < \infty$. The inner product is $(\psi, \psi') = \int (\psi(p)\psi'(p)) d\nu p$. (The integrand is a Borel function.) For $x \in J$ let $\bar{x}$ be the characteristic function of x, so that the set of all $\bar{x}$ is a complete orthonormal set for $L_2(\gamma)$. For each $\psi \in \Psi$ let $\psi_1(p, x) = (\psi(p), \bar{x})$ and observe (a small proof is needed) that ψ_1 is identically zero for x outside some countable subset of J, and that for each fixed x, the resulting function of p is a Borel function. Consequently, ψ_1 is measurable relative to $\nu \times \gamma$, and using the Fubini theorem, the $L_2(\nu \times \gamma)$ norm of ψ_1 is $\|\psi\|$. A straightforward proof shows that each member of $L_2(\nu \times \gamma)$ is obtained from some

member of Ψ in this way. Finally, for $f \in \mathcal{C}$ and $\psi \in \Psi$ set $(f\psi)(p) = f(p)\psi(p)$. In view of the preceding we have at once: if for $\psi \in \Psi$, $\psi_x(p) = (\psi(p), \bar{x})$ and $S\psi = \Sigma_1^1 \{\psi_x : x \in J\}$, then S is unitary and $S^{-1}T_fS\psi = f\psi$.

5. Re-assembly.—Given the spectrum X and the multiplicity ϕ, we describe, to a unitary equivalence, the Hilbert space H and the operator algebra $\mathfrak{A}$. We give three such descriptions, based on the three of the previous section. Because the proofs offer no new difficulty they are omitted. For convenience in statement we adopt the von Neumann definition of ordinal and cardinal numbers, so that each ordinal is identical with the set of all smaller ordinals, and a cardinal is an ordinal which cannot be put in 1-1 correspondence with a smaller ordinal. Let C be the supremum of the values of ϕ, and let ν be a restricted Borel measure on X, constructed from the characteristic measures as in section 2.

5.1. Let Θ be the set of functions on C to $L_2(\nu)$ such that for $c \in C$, $p \in X$, $\theta_c(P) = 0$ if $c \geqq \phi(p)$ and $\Sigma_1^1 \{\|\theta_c\|^2 : c \in C\} = \|\theta\|^2 < \infty$. For $f \in \mathcal{C}$ let $(f\theta)_a(p) = f(p)\theta_a(p)$. Then f on Θ is unitarily equivalent to T_f on H.

5.2. Let γ be counting measure on C, let K be the subset of $X \times C$ consisting of pairs (p, c) with $\phi(p) < c$, and let η be $\nu \times \gamma$ restricted to subsets of K. For $k \in L_2(\eta)$ and $f \in \mathcal{C}$ let $(fk)(p, c) = f(p)k(p, c)$. Then f on $L_2(\eta)$ is unitarily equivalent to T_f on H.

5.3. Let Ψ be the set of Borel functions ψ on X to $L_2(\gamma)$ such that ψ is zero outside of some restricted Borel set, the range of ψ is separable, and $\int \|\psi(p)\|^2 d\nu p = \|\psi\|^2 < \infty$. Let Ψ_0 be the set of members of Ψ such that for $c \in C$, $p \in X$, $\psi(p)(c) = 0$ if $c \geqq \phi(p)$. If for $f \in \mathcal{C}$, $(f\psi)(p) = f(p)\psi(p)$, then f on Ψ is unitarily equivalent to T_f on H. There is an alternate way of describing Ψ_0. For $p \in X$ let E_p be the projection of $L_2(\gamma)$ into itself obtained by multiplying each member by the characteristic function of the set of all $a, a < \phi(p)$. Let, for $\psi \in \Psi$, $(E\psi)(p) = E_p\psi(p)$. Then $\Psi_0 = \textit{range } E$. Observe that E has very special structure: for p and q in X either $E_p \geqq E_q$ or $E_q \geqq E_p$ and the cardinal of a complete orthonormal set for *range* E_p is $\phi(p)$.

* Presented, in part, to the International Congress of Mathematicians, September, 1950. This work was in part done under contract N7-onr-434, Task Order III, Navy Department, the Office of Naval Research.

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[2] Segal, I. E., "Decompositions of Operator Algebras I and II," Memoirs of the A.M.S. No. 9 (1951).

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ON THE POINCARÉ POLYNOMIAL OF THE FULL LINEAR GROUP

BY F. D. MURNAGHAN

INSTITUTO TECNÓLOGICO DE AERONÁUTICA, SÃO JOSÉ DOS CAMPOS, BRAZIL

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The Poincaré polynomial $P_n(z)$ of the full linear group of dimension n is furnished by the formula[1]

$$P_n(z) = \frac{(1+z)^n}{n!} \int_0^1 \ldots \int_0^1 \psi(z)\bar{\psi}(z)\Delta\bar{\Delta}d\phi_1 \ldots d\phi_n$$

where $\psi(z) = \prod_{i<k} (\epsilon_i + z\,\epsilon_k)$, $\epsilon_i = \exp(2\pi i\phi_k)$, $\Delta = \prod_{i<k} (\epsilon_i - \epsilon_k)$ and the superposed bars indicate complex conjugates. Weyl (*loc. cit.*) writes that "although one would hardly deem it difficult to evaluate the elementary integral (which furnishes $P_n(z)$) nobody so far has succeeded in doing this directly." The challenge in this statement has stood too long and we propose to meet it here. The idea which enabled us to do this came when studying an ingenious paper by Newell.[2]

We denote by $\sigma_0(=1)$, σ_1, ..., σ_n the elementary symmetric functions of the n quantities ϵ_1, ..., ϵ_n and by $\phi(t)$ the polynomial $\Pi(t+\epsilon_i)$ so that

$$\phi(t) = \sigma_0 t^n + \sigma_1 t^{n-1} + \ldots + \sigma_n.$$

Since $\bar{\epsilon}_i = 1/\epsilon_i$, $(1+z)^n\,\psi(z)\bar{\psi}(z)$ is the quotient of $\phi(z\,\epsilon_1)\ldots\phi(z\,\epsilon_n)$ by $(\epsilon_1\ldots\epsilon_n)^n$; furthermore $\int_0^1 \ldots \int_0^1 \psi(z)\bar{\psi}(z)\Delta\bar{\Delta}d\varphi_1\ldots d\phi_n$ is (by the theory of residues) the term independent of ϵ_1, ..., ϵ_n in $\psi(z)\bar{\psi}(z)\Delta\bar{\Delta}$ and $\bar{\Delta}$ is $(-1)^{n_2}\Delta$ divided by $(\epsilon_1\ldots\epsilon_n)^{n-1}$, where $n_2 = n(n-1)/2$, so that $P_n(z)$ is the product of the coefficient of $(\epsilon_1\ldots\epsilon_n)^{2n-1}$ in $\phi(z\,\epsilon_1)\ldots\phi(z\,\epsilon_n)\Delta^2$ by $(-1)^{n_2}/n!$ On denoting by A the $n \times 2n$ matrix

$$A = \begin{pmatrix} \sigma_0 & \sigma_1 \ldots & \sigma_n & 0 \ldots 0 \\ 0 & \sigma_0 \ldots & \sigma_{n-1} & \sigma_n \ldots 0 \\ \ldots & \ldots & \ldots & \ldots \\ 0 & 0 \ldots \sigma_0 & \sigma_1 & \ldots \sigma_n \end{pmatrix}$$

and by B the $2n \times n$ matrix

$$B = \begin{pmatrix} (z\,\epsilon_1)^{2n-1} & \ldots & (z\,\epsilon_n)^{2n-1} \\ (z\,\epsilon_1)^{2n-2} & \ldots & (z\,\epsilon_n)^{2n-1} \\ \cdot & & \cdot \\ \cdot & & \cdot \\ \cdot & & \cdot \\ z\,\epsilon_1 & \ldots & z\,\epsilon_n \\ 1 & \ldots & 1 \end{pmatrix}$$

we have

$$\det AB = \phi(z\,\epsilon_1)\ldots\phi(z\,\epsilon_n)z^{n_3}\Delta.$$

Now $\det AB$ is the sum of the products of corresponding n-rowed minors selected from A and B, respectively. The minor formed from the $(2n - l_1)$th, ..., $(2n - l_n)$th columns of A, where $l_1 > l_2 > \ldots > l_n \geqslant 0$, has $\sigma_{n-l_1}, \ldots, \sigma_{n-l_n}$ as the elements of its last row (it being understood that any σ carrying a negative subscript is zero) and so this minor is the character of the irreducible representation of the n-dimensional linear group which corresponds to the partition (μ) which is the associate of the partition $(\lambda) = (n - l_n, n + 1 - l_{n+1}\ldots, 2n - 1 - l_1)$. (NOTE: We have to use the associate partition (μ) rather than the partition (λ) since the elements of A are the elementary symmetric functions $\sigma_0, \ldots, \sigma_n$ and not the complete symmetric functions $p_0, p_1, \ldots$.) The product of this minor by Δ is, then, the alternant whose rows are the $(\mu_1 + n - 1)$th, $(\mu_2 + n - 2)$th, ..., μ_nth powers of $\epsilon_1, \ldots, \epsilon_n$. The corresponding minor of B has $z^{l_1+\cdots+l_n}$ as a factor, the remaining factor being the alternant whose rows are the l_1th, ..., l_nth powers of $\epsilon_1, \ldots, \epsilon_n$. The product of the two corresponding minors is, accordingly, the product of $z^{l_1+\cdots+l_n}$ by an n-rowed determinant whose elements are power sums of the $\epsilon_1, \ldots, \epsilon_n$. The first row of this determinant is $s(\mu_1 + n - 1 + l_1), \ldots, s(\mu_1 + n - 1 + l_n)$ where $s(k) = \epsilon_1^k + \ldots + \epsilon_n^k$. Similarly the second row is $s(\mu_2 + n - 2 + l_1), \ldots, s(\mu_2 + n - 2 + l_n)$ and so on. Since we are seeking the coefficient of $(\epsilon_1\ldots\epsilon_n)^{2n-1}$ we may neglect, in evaluating this n-rowed determinant, any $s(k)$ for which $k > 2n - 1$. For example, if $l_1 < 2n - 1$, the last element of $(\lambda) > 0$ so that $\mu_1 = n$ and we may neglect all the elements of the first row of our n-rowed determinant save when $l_n = 0$ (in which case we may neglect all elements of the first row save the last which is $s(2n - 1)$). Thus we need only consider those minors of A which contain either the first or the last column and we do not need to consider a minor of A which contains *both* the first and last columns; for in this case $l_1 = 2n - 1$, $l_n = 0$ and the first element of (λ) is n so that $\mu_n \geqslant 1$ and we may neglect all the elements of the first column of our n-rowed determinant (whose elements are power sums of $\epsilon_1, \ldots, \epsilon_n$).

Since the first column of A is $\begin{pmatrix} \sigma_0 \\ . \\ . \\ . \\ 0 \end{pmatrix}$ the contribution from the minors of A

which contain the first column of A is the product of $P_{n-1}(z)$ by z^{2n-1} (for we may remove the factor $\epsilon_1\ldots\epsilon_n$ from each corresponding minor of B

and neglect all elements of the first row of our n-rowed determinant, whose elements are power sums $s(k)$, save the last which is $s(2n-2)$); similarly, since the last column of A is $\begin{pmatrix} 0 \\ 0 \\ . \\ . \\ \sigma_n \end{pmatrix}$ the contribution from the minors of A which contain the last column of A is $P_{n-1}(z)$. Hence $P_n(z) = (z^{2n-1}+1)P_{n-1}(z)$ and, since $P_1(z) = z+1$, we obtain the well-known result $P_n(z) = (z+1)(z^3+1)\ldots(z^{2n-1}+1)$.

[1] Weyl, H., "The Classical Groups," p. 234. Princeton University Press, Princeton, N. J. (1939).

[2] Newell, M. J., *Proc. Lond. Math. Soc., Ser.* 2., **53**, 345–355 (1951).

ON THE POINCARÉ POLYNOMIALS OF THE CLASSICAL GROUPS

By F. D. Murnaghan

Instituto Tecnólogico de Aeronáutica, São José dos Campos, Brasil

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In a recent note in these PROCEEDINGS[1] we have obtained the Poincaré polynomial of the linear group of dimension n by direct evaluation of the integral which defines the polynomial. The method we used was somewhat specialized and we have been unable to successfully apply it to the determination of the Poincaré polynomials of the rotation and symplectic groups. We have since found, however, that the Poincaré polynomials of these various groups (the linear, the rotation and the symplectic) may be found by a method which involves hardly any calculation and we propose in the present note to indicate this method.

In the case of the linear group, of arbitrary dimension n, we may confine our attention to its unitary subgroup. If we denote by s a typical element of this unitary subgroup U and by $A(s)$ the corresponding element of the adjoint representation of U, the Poincaré polynomial of the n-dimensional linear group is (by definition) the integral of $\det(zE_n + A(s))$ over U. The adjoint representation $\{A(s)\}$ of U is n^2-dimensional, U being an n^2-parameter group, and it is reducible, being the sum of two irreducible components. Indeed $\{A(s)\}$ is the product of the self-representation $\{1\}$ of U by the representation obtained by dividing the matrices of the representation $\{1^{n-1}\}$ of U by $\det s$ and, since $\{1^{n-1}\}\{1\} = \{1^n\} + \{21^{n-2}\}$, it follows that $\{A(s)\}$ is the sum of the identity representation of U and the

irreducible representation, of dimension $n^2 - 1$, of U obtained by dividing the matrices of the representation $\{21^{n-2}\}$ of U by $\det s$. $\mathrm{Det}(zE_n + A(s))$ is a reciprocal polynomial of degree n^2, with highest coefficient 1, and the coefficient of z^{n^2-k} in this polynomial is $A(s) \otimes \{1^k\}$, $k = 0, 1, \ldots, n^2$. Since the integral of the character of any irreducible representation of U, other than the identity representation, over U is zero, while the integral of the character of the identity representation is 1, it follows that the coefficient of z^{n^2-k} in the Poincaré polynomial of the n-dimensional linear group is the number of times that $\{1^{n-1}\}\ \{1\} \otimes \{1^k\}$ contains the representation $\{k^n\}$ of U. The remarkable fact, which saves us the trouble of all but one relatively trivial calculation, is that this number is independent of n. In fact[2] $\{1^{n-1}\}\ \{1\} \otimes \{1^k\} = \Sigma_{(\alpha)}[\{1^{n-1}\} \otimes \{\alpha\}]\{\alpha^*\}$ where the sum is over all partitions (α) of k and (α^*) is the associate partition of k to (α). Since $\{1^{n-1}(s)\} = \det s\ \{1(s^{-1})\}$ it follows that $\{1^{n-1}\}\ \{1\} \otimes \{1^k\} = (\det s)^k\ \Sigma_{(\alpha)}\{\alpha(s^{-1})\}\ \{\alpha^*(s)\}$ which is independent of n. When $n = 1$ the Poincaré polynomial (of degree 1) is $z + 1$, since it is reciprocal and with highest coefficient 1. Hence, when $n = 2$, the Poincaré polynomial, of degree 4, is $z^4 + z^3 + * + z + 1$ the only unknown coefficient being that of z^2. This unknown coefficient is the number of times that $(\det s)^2[\{2(s^{-1})\}\ \{1^2(s)\} + \{1^2(s^{-1})\}\ \{2(s)\}]$ or, equivalently, $2\{2\}\ \{1^2\}$ involves $\{2^2\}$ and this is zero since $\{2\}\ \{1^2\} = \{31\} + \{21^2\}$. Thus, when $n = 2$, the Poincaré polynomial of the linear group is $z^4 + z^3 + z + 1 = (z^3 + 1)(z + 1)$. Hence, when $n = 3$, the Poincaré polynomial, of degree 9, is $(z^9 + z^8 + z^6 + z^5) + (z^4 + z^3 + z + 1)$ (there being no unknown coefficient) and this may be written as $(z^5 + 1)(z^3 + 1)(z + 1)$. Continuing this argument we see, without further calculation, that the Poincaré polynomial of the n-dimensional linear group is $(z^{2n-1} + 1)(z^{2n-3} + 1) \ldots (z^3 + 1)(z + 1)$.

The adjoint representation $\{A(s)\}$ of the n-dimensional rotation group is of dimension $n_2 = n(n-1)/2$, since the n-dimensional rotation group is an n_2-parameter group. It is, in fact, the representation $[1^2] = \{1^2\}$ so that, when $n = 2$, it is the identity representation (as it has to be since the 2-dimensional rotation group is a 1-parameter commutative group); when $n = 3$ it is the self-representation $\{1\}$ and, hence, irreducible; when $n = 4$, it is reducible, being the sum of two irreducible representations each of dimension 3; when $n > 4$ it is irreducible. By the same argument as in the case of the linear group the Poincaré polynomial of the n-dimensional rotation group is a reciprocal polynomial of degree n_2, with highest coefficient unity, the coefficient of z^{n_2-k} being the number of times that $\{1^2\} \otimes \{1^k\}$ contains the identity representation, $k = 0, 1, \ldots, n_2$. The analysis of $\{1^2\} \otimes \{1^k\}$ is known;[3, 4] thus $\{1^2\} \otimes \{1^2\} = \{21^2\}$; $\{1^2\} \otimes \{1^3\} = \{31^3\} + \{2^3\}$; $\{1^2\} \otimes \{1^4\} = \{41^4\} + \{32^21\}$; $\{1^2\} \otimes \{1^5\} = \{51^5\} + \{42^21^2\} + \{3^22^2\}$ and so on and we have merely to determine how often each term of these various analyses (of rep-

resentations of the linear group) contains the identity representation of the rotation subgroup. It is known[5] that the only irreducible representations $\{\lambda\}$ of the n-dimensional linear group which contain the identity representation $[0]$ of the rotation subgroup are those for which (λ) is a partition into even parts only, e.g., $(\lambda) = (0), (2), (4), (2^2), (6), (42), \ldots$ and that each of these contains $[0]$ precisely once. When $n = 2$ the Poincaré polynomial, being of the first degree, is $z + 1$; when $n = 3$, the Poincaré polynomial is of degree 3 and the coefficient of z^2 is zero since $\{1^2\}$ does not contain $[0]$. Hence, when $n = 3$, the Poincaré polynomial is $z^3 + 1$. When $n = 4$, the Poincaré polynomial is of degree 6 and is $z^6 + 0z^5 + 0z^4 + * + 0z^2 + 0z + 1$ and we have merely to determine the coefficient of z^3; since $\{1^2\} \otimes \{1^3\} = \{31^3\} + \{2^3\}$ and since, when $n = 4$, $\{31^3\} = \{2\}$ this coefficient is 2 so that the Poincaré polynomial is $z^6 + 2z^3 + 1$. Note that when n has any value other than 4, $\{1^2\} \otimes \{1^3\}$ contains $[0]$ once and not twice as is the case when $n = 4$ (for $\{31^3\}$ vanishes when $n < 4$ and is not a partition into even parts only when $n > 4$). When $n = 5$, the Poincaré polynomial, of degree 10, is $z^{10} + z^7 + z^3 + 1 = (z^7 + 1)(z^3 + 1)$; when $n = 6$, the Poincaré polynomial, of degree 15, is $z^{15} + z^{12} + z^{10} + z^8 + z^7 + z^5 + z^3 + 1 = (z^7 + 1)(z^5 + 1)(z^3 + 1)$ (the coefficient of z^{10} being 1 since $\{51^5\} = \{4\}$ when $n = 6$). Proceeding in this way we see that, when n is odd, the Poincaré polynomial of the n-dimensional rotation group is $(z^{2n-3} + 1)(z^{2n-7} + 1) \ldots (z^3 + 1)$ while, when n is even, the Poincaré polynomial of the n-dimensional rotation group is $(z^{2n-5} + 1)(z^{2n-9} + 1) \ldots (z^3 + 1)(z^{n-1} + 1)$.

The symplectic group is of even dimension $n = 2\nu$ and the adjoint representation is of dimension $n(n + 1)/2 = N$, say. It is the irreducible representation $\langle 2 \rangle = \{2\}$ and the Poincaré polynomial is a reciprocal polynomial of degree N, with highest coefficient 1, the coefficient of z^{N-k} being the number of times that the representation $\{2\} \otimes \{1^k\}$ of the n-dimensional linear group contains the identity representation $\langle 0 \rangle$ of its symplectic subgroup, $k = 0, 1, \ldots, N$. $\{2\} \otimes \{1^k\}$ is the associate of $\{1^2\} \otimes \{1^k\}$ so that $\{2\} \otimes \{1^2\} = \{31\}$, $\{2\} \otimes \{1^3\} = \{41^2\} + \{3^2\}$, $\{2\} \otimes \{1^4\} = \{51^3\} + \{431\}$, $\{2\} \otimes \{1^5\} = \{61^4\} + \{531^2\} + \{4^22\}$ and so on. It is known[6] that the only irreducible representations $\{\lambda\}$ of the n-dimensional linear group which contain the identity representation of the symplectic subgroup are those for which each part is repeated an even number of times, e.g., $\{1^2\}$, $\{2^2\}$, $\{1^4\}$, $\{3^2\}$, $\{2^21^2\}$ and so on. Thus, when $n = 2$, the Poincaré polynomial, of degree 3, is $z^3 + 1$; when $n = 4$, the Poincaré polynomial, of degree 10, is $z^{10} + z^7 + z^3 + 1 = (z^7 + 1)(z^3 + 1)$ and generally, the Poincaré polynomial of the symplectic group of (even) dimension n is $(z^{2n-1} + 1)(z^{2n-5} + 1) \ldots (z^3 + 1)$.

[1] Murnaghan, F. D., these PROCEEDINGS, **38**, 606–608 (1952).

[2] Littlewood, D. E., *Phil. Trans. Roy. Soc., Ser. A*, **239**, 331 (1944).

[3] Littlewood, D. E., "Theory of Group Characters," Oxford University Press, 1940, p. 238.

[4] Murnaghan, F. D., *Anais Acad. Bras. de Ciencias*, **23**, 17 (1951).

[5] *Loc. cit.*,[2] 393.

[6] *Loc. cit.*,[2] 395.

*AN INEQUALITY WITH ALTERNATING SIGNS**

By H. F. Weinberger

Institute for Fluid Dynamics and Applied Mathematics,
University of Maryland, College Park, Maryland

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Let $(a_1, \ldots, a_m)$ be m positive numbers arranged in descending order.

$$a_1 \geqslant a_2 \geqslant \ldots \geqslant a_m \geqslant 0. \tag{1}$$

We shall prove the following inequality, which was conjectured by L. E. Payne and A. Weinstein:

$$\sum_{k=1}^{m} (-1)^{k-1} a_k{}^r \geqslant \left[\sum_{k=1}^{m} (-1)^{k-1} a_k\right]^r \qquad \text{for } r > 1. \tag{2}$$

Most known inequalities are essentially concerned with positive terms. This inequality, however, has the interesting property of dealing with terms of alternating sign. It originated in a geometric problem in the theory of symmetrization, which will be briefly mentioned at the end of this paper. It appears likely that the inequality may have various other applications.

To prove (2), we let

$$F_m(a_1 \ldots a_m) = \sum_{k=1}^{m} (-1)^{k-1} a_k{}^r - \left[\sum_{k=1}^{m} (-1)^{k-1} a_k\right]^r \tag{3}$$

and prove that F_m is positive by induction. Note that

$$F_1(a_1) = 0 \tag{4}$$

and

$$F_2(a_1, a_2) = [a_1 - a_2 + a_2]^r - a_2{}^r - [a_1 - a_2]^r \geqslant 0 \qquad \text{for } r > 1 \tag{5}$$

by Minkowski's inequality, with equality holding if and only if $a_2 = 0$ or $a_1 = a_2$.

We now write

$$F_m(a_1, a_2 \ldots, a_m) = F_m(a_2, a_2, \ldots, a_m) + \int_{a_2}^{a_1} \frac{\partial F_m}{\partial a_1}(a_1, \ldots, a_m) da_1 \tag{6}$$

and note that

$$F_m(a_2, a_2, \ldots a_m) = F_{m-2}(a_3, \ldots a_m). \tag{7}$$

Finally,

$$\frac{\partial F_m}{\partial a_1}(a_1, \ldots, a_m) = r\left\{a_1^{r-1} - \left[\sum_1^m (-1)^{k-1}a_k\right]^{r-1}\right\} \geqq 0 \tag{8}$$

with equality holding only for $\sum_2^m (-1)^{k-1}a_k = 0$. Thus, if we assume that $F_{m-2} \geqslant 0$, we see from (6), (7), and (8) that

$$F_m(a_1, \ldots, a_m) \geqslant 0, \tag{9}$$

and our theorem is proved by induction. From the conditions needed for equality in (5) and (8) and from (1) we see that the equality in (2) will hold if and only if there is an odd number l such that all terms of the sum $\sum(-1)^{k-1}a_k$ preceding a_l cancel each other pairwise, as do all those following a_l, unless m is even in which case a_m must either be zero by itself or else all the terms of $\sum(-1)^{k-1}a_k$ must cancel pairwise.

As can be seen by putting $b_k = a_k^r$ and taking the rth root of both sides, the inequality (2) is reversed if $0 < r < 1$.

The geometric significance of (2) is seen by putting

$$r = p/q,\ p > q,\ p \text{ and } q \text{ positive integers}, \tag{10}$$

and

$$a_k = y_k^q. \tag{11}$$

It then becomes

$$\left[\sum_1^m (-1)^{k-1}y_k^p\right]^{1/p} \geqslant \left[\sum_1^m (-1)^{k-1}y_k^q\right]^{1/q}. \tag{12}$$

If the numbers y_k represent the radii of concentric spheres in a p-space, then the left-hand side of (12) is the radius of a single sphere having the total volume contained between the spheres of radius y_1 and y_2, y_3 and y_4, etc. The right-hand side of (12) is the equivalent radius in the same sense in a q-space. Inequality (12) states that this equivalent radius is monotone increasing with the dimension of the space. The condition for equality in (12) is simply that all the volume involved consist of a single sphere.

It should be noted that inequality (12) has a similar geometric application for any $p > q > 0$ in the generalized theory of symmetrization of Payne and Weinstein,† who consider bodies of revolution in a fictitious space of non-integral dimensions.

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† L. E. Payne and A. Weinstein, "Capacity, Virtual Mass, and Symmetrization." To appear in the *Pacific Journal of Mathematics*.

POLIOMYELITIS AND THE WEATHER

BY CHARLES ARMSTRONG, M.D.

NATIONAL INSTITUTES OF HEALTH, BETHESDA, MARYLAND

Read before The Academy, April 30, 1952

A recognized peculiarity of poliomyelitis, generally considered to be mainly contact borne, is the tendency for outbreaks to be confined in temperate zones to the warmer portions of the year.

This general correlation between the occurrence of poliomyelitis and high atmospheric temperature has long been recognized, but attempts to demonstrate a correlation between its incidence and atmospheric humidity have been without notable success. Such attempts, however, have usually considered relative humidity at atmospheric temperatures and thus, in reality, have treated man as though he were a cold-blooded animal and further have neglected the fact that in his upper respiratory tract man has a most remarkable and efficient air-conditioning apparatus—an apparatus which except under extreme atmospheric conditions warms inspired air to a uniform temperature of about 90°F. and adjusts its relative humidity to about 90% of saturation.

These temperature and humidity adjustments take place largely in the nose and throat.[1–3] Thus it necessarily follows that when cold, dry air of winter is breathed, increased secretions by the mucous membranes of the nose and throat must occur; otherwise, the membranes would soon be parched. Further it is apparent that in winter the secretions will be concentrated through the increased evaporation, and that these effects will result irrespective of whether the air is artificially warmed or not.

It is altogether probable that other mucous membrane variations accompany weather changes, such as altered blood supply, cellular reactions, etc. It is further obvious that the nearer atmospheric temperature approaches 90°F. the less does body temperature alter the drying effect of inspired air upon the mucous membranes.

These considerations lead one to suspect that an atmosphere which, as it is warmed to 90°F. upon inspiration, exerts a marked drying and concentrating effect upon the mucous secretions of the upper respiratory tract, tends to inhibit the spread of poliomyelitis, while an atmosphere which at 90°F. exerts little drying effect tends to favor such spread. Furthermore, it would appear that such effects upon the incidence of poliomyelitis might

result from direct contact of inspired air upon the membranes, provided the upper respiratory tract served either as a portal of entry or of exit for the poliomyelitis virus mainly effective in its spread from person to person; there is considerable field and laboratory evidence supporting the view that the nose and throat may serve in both capacities. Therefore, if these views are correct a correlation between the relative humidity of atmospheric air at a temperature of 90°F. and the occurrence of poliomyelitis should be apparent, provided a suitable area is selected for study.

A suitable area should have (1) A sufficiently prolonged and severe poliomyelitis outbreak to establish the existence of a susceptible population upon which modifying conditions might operate. (2) A limited area and relatively level contour. (3) A climate relatively free of frequent, sudden, and extreme atmospheric variations. (4) An alert medical profession and health department.

Let us now examine the poliomyelitis outbreak for such an area, the District of Columbia and Arlington County, Va., a relatively level area 10 miles square with 221 resident poliomyelitis cases in 1950.

Figure 1 shows the average atmospheric relative humidity at 90°F. and the incidence of poliomyelitis by date of onset for corresponding 7-day periods during 1950. Note that the epidemic began when the relative humidity reached 27–28% of saturation and disappeared when these levels and lower were reached in the fall.

A striking similarity is noted between the poliomyelitis and the humidity curves. For instance there are five major peaks on the humidity curve and five corresponding peaks on the poliomyelitis incidence curve, and the highest humidity peak is followed by the highest incidence peak. Furthermore, the lag between the corresponding peaks is uniformly three weeks in four instances and four weeks in one. During the first week of this 4-week interval (points E and E') the humidity was below 27% and the incidence actually fell, thus possibly explaining the longer period of lag in this instance.

Outbreak in a Semiarid Region.—Since a moist atmosphere at 90°F. apparently accompanies the height of the epidemic and a dry atmosphere to be associated with its disappearance, let us consider the outbreaks at Denver, Colo., 1950 and 1951, to see whether the occurrence of poliomyelitis in this semiarid region, average annual rainfall 13.99 inches, is compatible with the above suggested relationship.

Figure 2 shows the average atmospheric temperature, the average relative humidity at 90°F., and the number of poliomyelitis cases and the rainfall, all by months (1950). Note that the humidity curve rises to two peaks, each followed by a peak on the poliomyelitis curve, and that the depression in the latter follows the depression in the humidity curve corresponding to a very dry August. The total number of cases for the year was 69.

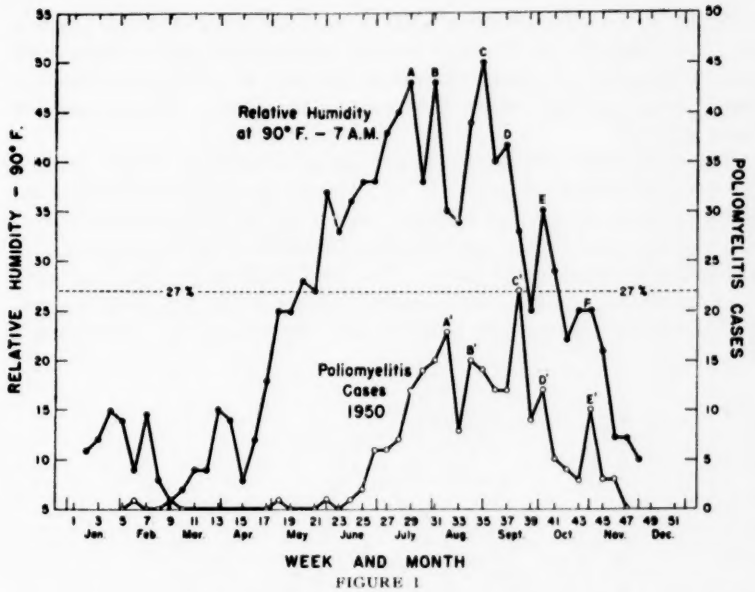

FIGURE 1

Average relative humidity by weeks: 7 A.M. readings adjusted to 90°F., at National Airport, Va., 1950. Poliomyelitis cases given for District of Columbia and Arlington County, Va., by week of onset.

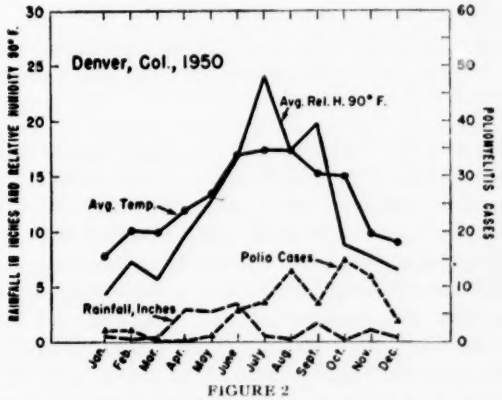

FIGURE 2

Average atmospheric temperature, average humidity of atmosphere at 90°F., precipitation, and poliomyelitis cases, by months. Denver, 1950.

Figure 3 shows the same type of curves for 1951. On August 2, 3, and 4 an exceptionally heavy rain of $3^1/_2$ inches occurred, and the accompanying high relative humidity was followed by a rapid rise in poliomyelitis incidence, which in turn fell with the drop in humidity. Total number of cases was 242.

The 69 cases for 1950 are too few to justify a study by weeks, but one has been attempted for 1951 (Fig. 4). Figure 3 reveals a noticeable similarity between the average humidity curves and the poliomyelitis curve, and the high peak on the humidity curve is followed by the high peak on the poliomyelitis incidence curve. The large variations in the four daily readings which enter into the averages, however, render the area unsuited for tracing fine relationships. For instance, the epidemic began and ended

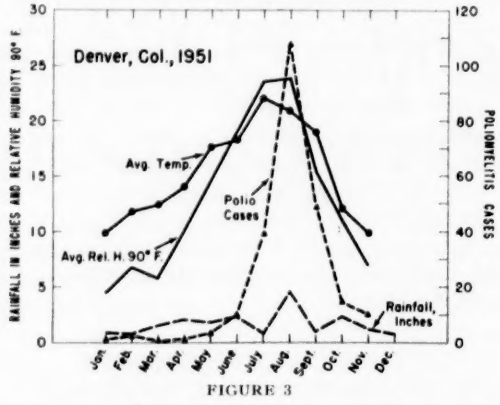

FIGURE 3

Average atmospheric temperature, average humidity of atmosphere at 90°F., precipitation, and poliomyelitis cases, by months. Denver, 1951.

when the average weekly humidity of 90°F., based on four daily readings, reached the neighborhood of 16% of saturation. If, however, we consider the average of the maximum daily readings for the week, the figure becomes 21%; and if the maximum single reading for each week is considered, the epidemic began and ended with a humidity of 27–30%.

Moreover, in Denver, elevation 5292 feet, it is possible for people traveling relatively short distances to descend a few thousand feet and thus encounter climatic variations such as might be encountered in a plains area by traveling several hundred miles to the south. It is, of course, not possible to determine how many, if any, Denver cases have contracted their poliomyelitis at lower elevations where, just as in the south, poliomyelitis may be expected to begin earlier and to persist later.

At any rate the general similarity between the curves is so apparent as to suggest no incompatibility.

Summary.—It is felt that the relative humidity of atmospheric air at 90°F. is of more importance in the spread of poliomyelitis than is atmos-

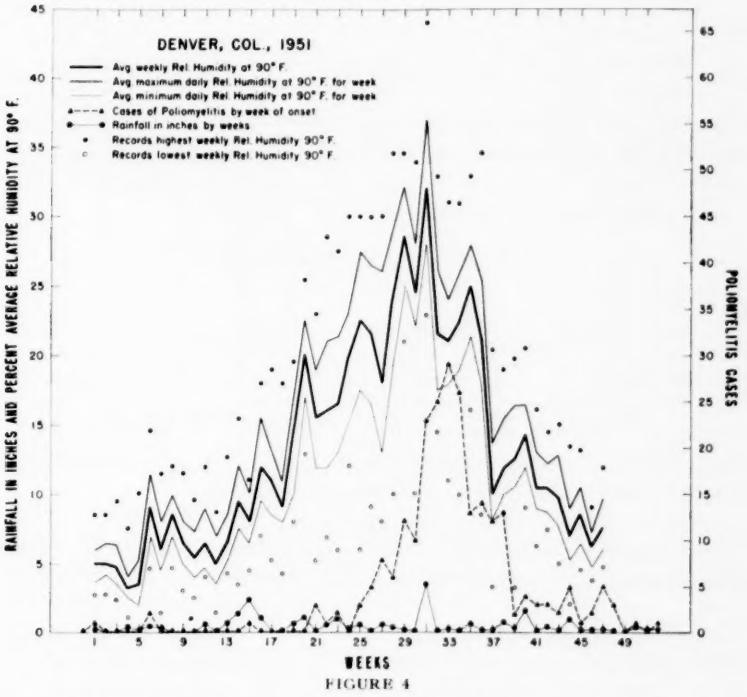
DENVER, COL., 1951
Avg. weekly Rel. Humidity at 90° F.
Avg. maximum daily Rel. Humidity at 90° F. for week
Avg. minimum daily Rel. Humidity at 90° F. for week
Cases of Poliomyelitis by week of onset
Rainfall in inches by weeks
Records highest weekly Rel. Humidity 90° F.
Records lowest weekly Rel. Humidity 90° F.
RAINFALL IN INCHES AND PERCENT AVERAGE RELATIVE HUMIDITY AT 90° F.
POLIOMYELITIS CASES
WEEKS
0 5 10 15 20 25 30 35 40 45
0 5 10 15 20 25 30 35 40 45 50 55 60 65
1 5 9 13 17 21 25 29 33 37 41 45 49

FIGURE 4

Average atmospheric humidity (4 daily readings) at 90° F.; average maximum atmospheric humidity (highest daily reading) at 90° F.; average minimum atmospheric humidity (lowest daily reading) at 90°F.; precipitation; and poliomyelitis cases, by weeks.

pheric temperature *per se*, and that the evidence points to the upper respiratory tract of man as a significant area in determining the spread of poliomyelitis. It further appears possible to predict, for a suitable limited area, the probable course of a poliomyelitis outbreak from two to three weeks in advance.

[1] Kayser, R., "Die Bedeutung der Nase und der Ersten Atmung fur die Respiration," *Arch. Ges. Physiol.*, **41**, 127–147 (1887).

[2] Seeley, L. E., "Study of Changes in Temperature and Water Vapor Content of

Respired Air in the Nasal Cavity," *Heating, Piping, and Air Conditioning*, **12**, 377–388 (1940).

[3] Winslow, C. E. A., Herrington, L. P., and Nelbach, J. H., "The Influence of Atmospheric Temperature and Humidity Upon the Dryness of the Oral Mucosa," *Am. J. Hyg.*, **35**, 27–39 (1942).

ERRATA

In the paper "Some Problems Involving Primitive Roots in a Finite Field," these PROCEEDINGS, **38**, 314 (1952), Theorem 13 on p. 318 should read

The number of solutions $N_{1,2s}(\alpha)$ *of*

$$\alpha = \gamma\beta + \delta_1\xi_1^2 + \ldots + \delta_{2s}\xi_{2s}^2 \qquad (\gamma\delta_i \neq 0)$$

is determined by

$$N_{1,2s}(0) = (p^{n(2s-1)} - p^{n(s-1)}\psi(\zeta))\phi(e),$$

and for $\alpha \neq 0$

$$N_{1,2s}(\alpha) = (p^{n(2s-1)} - p^{n(s-1)}\psi(\zeta))\phi(e) + p^{ns}\psi(\zeta)\omega(\alpha),$$

where $\psi(\zeta) = +1$ or -1 *according as* $\zeta = (-1)^s\delta_1 \ldots \delta_{2s}$ *is or is not a square of* $GF(p^n)$, *and* $\omega(\alpha)$ *has the same meaning as in Lemma 2.*

In the paper "The Number of Solutions of Certain Equations in a Finite Field," these PROCEEDINGS, **38**, 515 (1952), Theorem 10 on page 518 should read

Let $(a_{i1}, \ldots, a_{ik_i}) = d_i$, $(d_i, d_j) = 1$ *for* $i \neq j$. *Then the number of solutions of*

$$\sum_{i=1}^{t} \alpha_i \prod_{j=1}^{k_i} \xi_{ij}^{a_{ij}} = 0, \tag{5.1}$$

with $\Pi_{i,j}\,\xi_{ij} \neq 0$, *is*

$$q^{-1}(q-1)^{k-t+1}((q-1)^{t-1} + (-1)^t) \qquad (k = k_1 + \ldots + k_t).$$

The total number of solutions of (5.1) *is* q^{k-1}.

L. CARLITZ

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Abbott, Charles **G**reeley, 1915 (2), Smithsonian Institution, Washington 25, D. C.

Adams, Comfort **A**very, 1930 (4), 417 West Price Street, Philadelphia 44, Pennsylvania

Adams, Leason **H**eberling, 1943 (13), Carnegie Institution of Washington, 1530 P Street, N. W., Washington 5, D. C.

Adams, Roger, 1929 (5), University of Illinois, Urbana, Illinois

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* Members of the Executive Committee of the Council of the Academy.

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Adams, Walter Sydney, 1917 (2), Mount Wilson Observatory, Pasadena 4, California

Albert, Abraham **Adrian,** 1943 (1), Department of Mathematics, University of Chicago, Chicago 37, Illinois

Albright, Fuller, 1952 (10), Massachusetts General Hospital, Boston 14, Massachusetts

Alexander, James Waddell, 1930 (1), 29 Cleveland Lane, Princeton, New Jersey

Allee, Warder **C**lyde, 1951 (8), Department of Biology, University of Florida, Gainesville, Florida

Allen, Eugene Thomas, 1930 (6), The Irvington, 135 Pleasant Street, Arlington 74, Massachusetts

Allison, Samuel King, 1946 (3), Institute for Nuclear Studies, University of Chicago, Chicago 37, Illinois

Alvarez, Luis Walter, 1947 (3), Radiation Laboratory, University of California, Berkeley 4, California

Anderson, Carl David, 1938 (3), California Institute of Technology, Pasadena 4, California

Anderson, Rudolph John, 1946 (9), Department of Chemistry, Yale University, New Haven, Connecticut

Armstrong, Charles, 1944 (10), National Institutes of Health, Bethesda 14, Maryland

Avery, Oswald Theodore, 1933 (10), Hoods Hill Road, Nashville 5, Tennessee

Babcock, Ernest Brown, 1946 (7), Department of Genetics, University of California, Berkeley 4, California

Babcock, Harold Delos, 1933 (2), Mount Wilson Observatory, Pasadena 4, California

Bacher, Robert Fox, 1947 (3), California Institute of Technology, Pasadena 4, California

Badger, Richard McLean, 1952 (5), Gates and Crellin Laboratories of Chemistry, California Institute of Technology, Pasadena 4, California

Bailey, Irving Widmer, 1929 (7), Biological Laboratories, Harvard University, Cambridge 38, Massachusetts

Bailey, Liberty **H**yde, 1917 (7), Bailey Hortorium, Ithaca, New York

Bainbridge, Kenneth Tompkins, 1946 (3), Department of Physics, Harvard University, Cambridge 38, Massachusetts

Ball, Eric Glendinning, 1948 (9), Department of Biological Chemistry, Harvard University Medical School, Boston 15, Massachusetts

Bancroft, Wilder Dwight, 1920 (5), 7 East Avenue, Ithaca, New York

Bard, Philip, 1944 (9), Johns Hopkins Medical School, 710 North Washington Street, Baltimore 5, Maryland

Bartelmez, George William, 1949 (8), Department of Embryology, Carnegie Institution of Washington, Wolfe and Madison Streets, Baltimore 5, Maryland

Bartlett, Paul Doughty, 1947 (5), Harvard University, 12 Oxford Street, Cambridge 38, Massachusetts

Baxter, Gregory Paul, 1916 (5), T. Jefferson Coolidge, Jr. Memorial Laboratory, Harvard University, Cambridge 38, Massachusetts

Beach, Frank Ambrose, 1949 (12), Department of Psychology, Yale University, 333 Cedar Street, New Haven 11, Connecticut

Beadle, George Wells, 1944 (7), Department of Biology, California Institute of Technology, Pasadena 4, California

Beams, Jesse Wakefield, 1943 (3), Rouss Physical Laboratory, University of Virginia, Charlottesville, Virginia

Bell, Eric Temple, 1927 (1), California Institute of Technology, Pasadena 4, California

Berkey, Charles Peter, 1927 (6), Department of Geology, Columbia University, New York 27, New York

Berkner, Lloyd Viel, 1948 (13), Associated Universities, Inc., Room 6920, 350 Fifth Avenue, New York 1, New York

Bethe, Hans Albrecht, 1944 (3), Cornell University, Ithaca, New York

Bigelow, Henry Bryant, 1931 (8), Museum of Comparative Zoology, Harvard University, Cambridge 38, Massachusetts

Birch, Albert **Francis,** 1950 (6), Department of Geology, Harvard University, Cambridge 38, Massachusetts

Birge, Raymond Thayer, 1932 (3), University of California, Berkeley 4, California

Bjerknes, Jacob, 1947 (13), Department of Meteorology, University of California, Los Angeles 24, California

Blackwelder, Eliot, 1936 (6), P. O. Box N, Stanford University, Stanford, California

Blakeslee, Albert Francis, 1929 (7), Genetics Experiment Station, Smith College, Northampton, Massachusetts

Blalock, Alfred, 1945 (10), Johns Hopkins Hospital, Baltimore 5, Maryland

Bloch, Felix, 1948 (3), Department of Physics, Stanford University, Stanford, California

Bochner, Salomon, 1950 (1), Department of Mathematics, Princeton University, Princeton, New Jersey

Bogert, Marston Taylor, 1916 (5), 1158 Fifth Avenue, Apt. 14B, New York 29, New York

Bolton, Elmer K., 1946 (5), 2310 West 11th Street, Wilmington, Delaware

Bonner, James Frederick, 1950 (7), Kerckhoff Laboratories of Biology, California Institute of Technology, Pasadena 4, California

Boring, Edwin Garrigues, 1932 (12), Memorial Hall, Harvard University, Cambridge 38, Massachusetts

Bowen, Ira Sprague, 1936 (2), Mount Wilson Observatory, Pasadena 4, California

Bowen, Norman Levi, 1935 (6), Geophysical Laboratory, Carnegie Institution of Washington, 2801 Upton Street, N. W., Washington 8, D. C.

Bradbury, Norris Edwin, 1951 (3), Los Alamos Scientific Laboratory, P. O. Box 1663, Los Alamos, New Mexico

Bradley, Wilmot Hyde, 1946 (6), United States Geological Survey, Washington 25, D. C.

Breit, Gregory, 1939 (3), Sloane Physics Laboratory, Yale University, New Haven, Connecticut

Bridgman, Percy Williams, 1918 (3), Lyman Laboratory of Physics, Harvard University, Cambridge 38, Massachusetts

Briggs, Lyman James, 1942 (4), National Bureau of Standards, Washington 25, D. C.

Brink, Royal Alexander, 1947 (7), Department of Genetics, University of Wisconsin, Madison 6, Wisconsin.

Brode, Robert Bigham, 1949 (3), Department of Physics, University of California, Berkeley 4, California

Bronk, Detlev Wulf, 1939 (9), Johns Hopkins University, Baltimore 18, Maryland

Brouwer, Dirk, 1951 (2), Yale University Observatory, Prospect and Canner Streets, New Haven 11, Connecticut

Bucher, Walter Hermann, 1938 (6), Department of Geology, Columbia University, New York 27, New York

Buckley, Oliver Ellsworth, 1937 (4), Bell Telephone Laboratories, 463 West Street, New York 14, N. Y.

Buddington, Arthur Francis, 1943 (6), Princeton University, Princeton, New Jersey

Burkholder, Paul Rufus, 1949 (7), Osborn Botanical Laboratory, Yale University, New Haven 11, Connecticut

Bush, Vannevar, 1934 (4), Carnegie Institution of Washington, Washington 5, D. C.

Byerly, Perry, 1946 (13), Department of Seismology, University of California, Berkeley 4, California

Byers, Horace Robert, 1952 (13), Department of Meteorology, University of Chicago, Chicago 37, Illinois

Cannon, Paul Roberts, 1946 (10), Department of Pathology, University of Chicago, Chicago 37, Illinois

Carlson, Anton Julius, 1920 (9), University of Chicago, Chicago 37, Illinois

Carmichael, Leonard, 1943 (12), Tufts College, Medford 55, Massachusetts

Castle, William Bosworth, 1939 (10), Boston City Hospital, Boston 18, Massachusetts

Castle, William Ernest, 1915 (8), 421 Spruce Street, Berkeley 8, California

Chandler, William Henry, 1943 (7), College of Agriculture, University of California, 405 Hilgard Avenue, Los Angeles 24, California

Chaney, Ralph Works, 1947 (6), Department of Paleontology, University of California, Berkeley 4, California

Chevalley, Claude, 1952 (1), 468 Riverside Drive, New York 27, New York

Clark, William Mansfield, 1928 (9), Johns Hopkins Medical School, 710 North Washington Street, Baltimore 5, Maryland

Clarke, Hans Thacher, 1942 (9), United States Embassy, London, England

Clausen, Roy Elwood, 1951 (7), Division of Genetics, University of California, Berkeley 4, California

Cleland, Ralph Erskine, 1942 (7), Indiana University, Bloomington, Indiana

Clemence, Gerald Maurice, 1952 (2), U. S. Naval Observatory, Washington 25, D. C.

Cleveland, Lemuel Roscoe, 1952 (8), The Biological Laboratories, Harvard University, Cambridge 38, Massachusetts

Cloos, Ernst, 1950 (6), Department of Geology, Johns Hopkins University, Baltimore 18, Maryland

Coble, Arthur Byron, 1924 (1), 1907 Tondolea Lane, La Canada, California

Coblentz, William Weber, 1930 (3), 2737 Macomb Street, N. W., Washington 8, D. C.

Cochrane, Edward Lull, 1945 (4), The Westchester, Apt. 829 B, 4000 Cathedral Avenue, Washington 16, D. C.

Coggeshall, Lowell Thelwell, 1949 (10), Division of Biological Sciences, University of Chicago, Chicago 37, Illinois

Cohn, Edwin Joseph, 1943 (9), Harvard Medical School, Boston 15, Massachusetts

Cole, Rufus, 1922 (10), Mt. Kisco, New York

Compton, Arthur Holly, 1927 (3), Washington University, St. Louis 5, Missouri

Compton, Karl Taylor, 1924 (3), Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Conant, James Bryant, 1929 (5), Harvard University, Cambridge 38, Massachusetts

Condon, Edward Uhler, 1944 (3), 140 High Road, Corning, New York

Conklin, Edwin Grant, 1908 (8), Princeton University, Princeton, New Jersey

Coolidge, William David, 1925 (3), 1480 Lenox Road, Schenectady, New York

Cope, Arthur Clay, 1947 (5), Department of Chemistry, Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Cori, Carl Ferdinand, 1940 (9), School of Medicine, Washington University, Euclid Avenue and Kingshighway, St. Louis 10, Missouri

Cori, Gerty Theresa, 1948 (9), Department of Biological Chemistry, School of Medicine, Washington University, Euclid Avenue and Kingshighway, St. Louis 10, Missouri

Corner, George Washington, 1940 (8), Department of Embryology, Carnegie Institution of Washington, Wolfe and Madison Streets, Baltimore 5, Maryland. Academic year 1952–1953: 8, Merton Street, Flat B, Oxford, England

Couch, John Nathaniel, 1943 (7), University of North Carolina, Chapel Hill, North Carolina

Craig, Lyman Creighton, 1950 (5), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Crew, Henry, 1909 (3), 620 Library Place, Evanston, Illinois

Curme, George Oliver, Jr., 1944 (4), Carbide and Carbon Chemicals Corporation, 30 East 42nd Street, New York 11, New York

Daly, Reginald Aldworth, 1925 (6), 23 Hawthorn Street, Cambridge 38, Massachusetts

Danforth, Charles Haskell, 1942 (8), Department of Anatomy, Stanford University, Stanford University, California

Daniels, Farrington, 1947 (5), Department of Chemistry, University of Wisconsin, Madison 6, Wisconsin

Davis, Bergen, 1929 (3), 44 Morningside Drive, New York 25, New York

Davis, Hallowell, 1948 (9), Central Institute for the Deaf, 818 South Kingshighway, St. Louis 10, Missouri

Davisson, Clinton Joseph, 1929 (3), 2605 Jefferson Park Circle, Charlottesville, Virginia

Day, Arthur Louis, 1911 (6), 9113 Old Georgetown Road, Bethesda 14, Maryland

Debye, Peter, (1931) 1947* (5), Baker Laboratory, Cornell University, Ithaca, New York

DeGolyer, Everette Lee, 1951 (6), 1000 Continental Building, Dallas 1, Texas

Delbrück, Max, 1949 (7), Kerckhoff Laboratories of Biology, California Institute of Technology, Pasadena 4, California

Demerec, Milislav, 1946 (8), Department of Genetics, Carnegie Institution of Washington, Cold Spring Harbor, New York

Detwiler, Samuel Randall, 1932 (8), College of Physicians and Surgeons, 630 West 168th Street, New York 32, New York

Dobzhansky, Theodosius, 1943 (8), Department of Zoology, Columbia University, New York 27, New York

Dochez, Alphonse Raymond, 1933 (10), Columbia University, Presbyterian Hospital, 620 West 168th Street, New York 32, New York

Dodge, Bernard Ogilvie, 1933 (7), New York Botanical Garden, Bronx Park (Fordham Station), New York 58, New York

Doisy, Edward Adelbert, 1938 (9), St. Louis University School of Medicine, 1402 South Grand Boulevard, St. Louis 4, Missouri

Douglas, Jesse, 1946 (1), 2610 Glenwood Road, Brooklyn 10, New York

Dragstedt, Lester Reynold, 1950 (10), Department of Surgery, University of Chicago, 950 East 59th Street, Chicago 37, Illinois

Dryden, Hugh Latimer, 1944 (4), National Advisory Committee for Aeronautics, 1724 F Street, N. W., Washington 25, D. C.

DuBois, Eugene Floyd, 1933 (9), Cornell University Medical College, 1300 York Avenue, New York 21, New York

Dubos, René Jules, 1941 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

DuBridge, Lee Alvin, 1943 (3), California Institute of Technology, Pasadena 4, California

Duggar, Benjamin Minge, 1927 (7), Lederle Laboratories, Incorporated, Pearl River, New York

* Elected a foreign associate in 1931; became a naturalized citizen in 1946 and a member of the Academy in 1947.

Dunbar, Carl Owen, 1944 (6), Peabody Museum, Yale University, New Haven, Connecticut

Dunn, Gano, 1919 (4), 80 Broad Street, New York 4, New York

Dunn, Leslie Clarence, 1943 (8), Columbia University, New York 27, New York

Dunning, John Ray, 1948 (3), Department of Physics, Columbia University, New York 27, New York

Durand, William Frederick, 1917 (4), Mohawk Hotel, 379 Washington Avenue, Brooklyn, New York

du Vigneaud, Vincent, 1944 (9), Cornell University Medical College, New York 21, New York

Edsall, John Tileston, 1951 (5), Laboratory of Physical Chemistry, Harvard University, 25 Shattuck Street, Boston 15, Massachusetts

Einstein, Albert (1922) 1942† (3), The Institute for Advanced Study, Princeton, New Jersey

Eisenhart, Luther Pfahler, 1922 (1), 25 Alexander Street, Princeton, New Jersey

Elderfield, Robert Cooley, 1949 (5), Department of Chemistry, University of Michigan, Ann Arbor, Michigan

Elvehjem, Conrad Arnold, 1942 (9), Department of Biochemistry, University of Wisconsin, Madison 6, Wisconsin

Epstein, Paul Sophus, 1930 (3), 1484 Oakdale Street, Pasadena 4, California

Erlanger, Joseph, 1922 (9), Washington University School of Medicine, 4580 Scott Avenue, St. Louis 10, Missouri

Evans, Griffith Conrad, 1933 (1), Department of Mathematics, University of California, Berkeley 4, California

Evans, Herbert McLean, 1927 (9), Institute of Experimental Biology, University of California, Berkeley 4, California

Ewing, William **Maurice,** 1948 (13), Columbia University, Lamont Geological Observatory, Torrey Cliff, Palisades, New York

Eyring, Henry, 1945 (5), Graduate School, University of Utah, Salt Lake City, Utah

Fenn, Wallace Osgood, 1943 (9), School of Medicine and Dentistry, University of Rochester, 260 Crittenden Boulevard, Rochester 7, New York

Fermi, Enrico, 1945 (3), Institute for Nuclear Studies, University of Chicago, Chicago 37, Illinois

Fieser, Louis Frederick, 1940 (5), Harvard University, Cambridge 38, Massachusetts

Fleming, John Adam, 1938 (13), Carnegie Institution of Washington, 1530 P Street, N. W., Washington 5, D. C.

Fletcher, Harvey, 1935 (4), Department of Electrical Engineering, Columbia University, New York 27, New York

Folkers, Karl August, 1948 (5), Research and Development Division, Merck and Company, Incorporated, Rahway, New Jersey

† Elected a foreign associate in 1922; became a naturalized citizen in 1941 and a member of the Academy in 1942.

Foote, Paul Darwin, 1943 (4), Gulf Research and Development Company, P. O. Drawer 2038, Pittsburgh 30, Pennsylvania

Forbes, Alexander, 1936 (9), Harland Street, Milton, Massachusetts

Francis, Thomas, Jr., 1948 (10), Department of Epidemiology, School of Public Health, University of Michigan, Ann Arbor, Michigan

Franck, James, 1944 (3), Institute of Radiobiology and Biophysics, University of Chicago, Chicago 37, Illinois

Fred, Edwin Broun, 1931 (7), University of Wisconsin, Madison 6, Wisconsin

Fruton, Joseph Stewart, 1952 (9), Department of Physiological Chemistry, Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut

Fuoss, Raymond Matthew, 1951 (5), Sterling Chemistry Laboratory, Yale University, 225 Prospect Street, New Haven 11, Connecticut

Fuson, Reynold Clayton, 1944 (5), 263 Noyes Laboratory, University of Illinois, Urbana, Illinois

Gamble, James Lawder, 1945 (10), 33 Edge Hill Road, Brookline, Massachusetts

Gasser, Herbert Spencer, 1934 (9), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Gesell, Arnold, 1947 (12), Clinic of Child Development, Yale University School of Medicine, 14 Davenport Avenue, New Haven, Connecticut

Giauque, William Francis, 1936 (5), University of California, Berkeley 4, California

Gibbs, William Francis, 1949 (4), One Broadway, New York, New York

Gilliland, Edwin Richard, 1948 (4), Department of Chemical Engineering, Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Gilluly, James, 1947 (6), U. S. Geological Survey, Denver Federal Center, Denver 14, Colorado

Gilman, Henry, 1945 (5), Department of Chemistry, Iowa State College, Ames, Iowa

Goddard, David Rockwell, 1950 (7), Botanical Laboratory, University of Pennsylvania, 38th and Woodland Avenue, Philadelphia 4, Pennsylvania

Goldschmidt, Richard Benedikt, 1947 (8), Department of Zoology, University of California, Berkeley 4, California

Goodpasture, Ernest William, 1937 (10), Vanderbilt University, Nashville 4, Tennessee

Goudsmit, Samuel Abraham, 1947 (3), Theoretical Physics Division, Brookhaven National Laboratory, Upton, Long Island, New York

Graham, Clarence Henry, 1946 (12), Department of Psychology, Columbia University, New York 27, New York

Graham, Evarts Ambrose, 1941 (10), Barnes Hospital, 600 South Kingshighway, St. Louis 10, Missouri

Greenewalt, Crawford Hallock, 1952 (4), E. I. du Pont de Nemours and Company, Incorporated, Wilmington, Delaware

Gregory, William King, 1927 (6), American Museum of Natural History, 77th Street and Central Park West, New York 24, New York

Griggs, David Tressel, 1952 (13), Office of the Chief of Staff, United States Air Force, Washington 25, D. C.

Gunn, Ross, 1951 (13), Weather Bureau, U. S. Department of Commerce, Washington 25, D. C.

Gutenberg, Beno, 1945 (13), Seismological Laboratory, 220 North San Rafael Avenue, Pasadena 2, California

Hammett, Louis Plack, 1943 (5), Department of Chemistry, Columbia University, New York 27, New York

Harlow, Harry F., 1951 (12), Bascom Hall, University of Wisconsin, Madison 6, Wisconsin

Harned, Herbert Spencer, 1950 (5), Sterling Chemistry Laboratory, Yale University, 225 Prospect Street, New Haven 11, Connecticut

Harrison, Ross Granville, 1913 (8), Osborn Zoological Laboratory, Yale University, New Haven, Connecticut

Hart, Edwin Bret, 1944 (9), Biochemistry Building, University of Wisconsin, Madison 6, Wisconsin

Hartline, Haldan Keffer, 1948 (9), Department of Biophysics, Biological Laboratories, Johns Hopkins University, Baltimore 18, Maryland

Hartman, Carl Gottfried, 1937 (8), Ortho Research Foundation, Raritan, New Jersey

Harvey, Edmund Newton, 1934 (8), Guyot Hall, Princeton University, Princeton, New Jersey

Hastings, Albert Baird, 1939 (9), Department of Biological Chemistry, Harvard University Medical School, Boston 15, Massachusetts

Heidelberger, Michael, 1942 (10), College of Physicians and Surgeons, 620 West 168th Street, New York 32, New York

Hendricks, Sterling Brown, 1952 (5), Bureau of Plant Industry, U. S. Department of Agriculture, Beltsville, Maryland

Herrick, Charles Judson, 1918 (8), 236 Morningside Drive, S. E., Grand Rapids, Michigan

Herty, Charles Holmes, Jr., 1947 (4), Bethlehem Steel Company, Incorporated, Bethlehem, Pennsylvania

Hess, Harry Hammond, 1952 (6), Department of Geology, Princeton University, Princeton, New Jersey

Hewett, Donnel Foster, 1937 (6), 1460 Rose Villa Street, Pasadena 4, California

Hildebrand, Joel Henry, 1929 (5), Gilman Hall, University of California, Berkeley 4, California

Hilgard, Ernest Ropiequet, 1948 (12), Department of Psychology, Stanford University, Stanford, California

Hisaw, Frederick Lee, 1947 (8), Biological Laboratories, Harvard University, 16 Divinity Avenue, Cambridge 38, Massachusetts

Hooton, Earnest Albert, 1935 (11), Peabody Museum, Harvard University, Cambridge 38, Massachusetts

Hoover, Herbert Clark, 1922 (4), Waldorf Astoria Towers, New York, New York

Horsfall, Frank Lappin, **Jr.,** 1948 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Houston, William Vermillion, 1943 (3), The Rice Institute, Houston, Texas

Hubble, Edwin Powell, 1927 (2), Mount Wilson Observatory, Pasadena 4, California

Hubbs, Carl Leavitt, 1952 (8), Scripps Institution of Oceanography, La Jolla, California

Hudson, Claude Silbert, 1927 (5), National Institutes of Health, Bethesda 14, Maryland

Huggins, Charles Brenton, 1949 (10), Department of Surgery, University of Chicago, 950 East 59th Street, Chicago 37, Illinois

Hull, Albert Wallace, 1929 (3), Research Laboratory, General Electric Company, Schenectady 5, New York

Hunsaker, Jerome Clark, 1935 (4), Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Hunter, Walter Samuel, 1935 (12), Brown University, Providence 12, Rhode Island

Hutchinson, George Evelyn, 1950 (8), Osborn Zoological Laboratory, Yale University, New Haven 11, Connecticut

Ipatieff, Vladimir **N**ikolaevich, 1939 (5), Universal Oil Products Company, Riverside, Illinois

Irwin, Malcolm **R**obert, 1950 (8), Department of Genetics, University of Wisconsin, Madison 6, Wisconsin

Iselin, Columbus **O'D**onnell, 1951 (13), Woods Hole Oceanographic Institution, Woods Hole, Massachusetts

Ives, Herbert Eugene, 1933 (3), 32 Laurel Place, Upper Montclair, New Jersey

Jacobs, Merkel **H**enry, 1939 (8), School of Medicine, University of Pennsylvania, Philadelphia 4, Pennsylvania

Jacobs, Walter Abraham, 1932 (5), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Jeffries, Zay, 1939 (4), General Electric Company, 1 Plastics Avenue, Pittsfield, Massachusetts

Johnson, John Raven, 1948 (5), Department of Chemistry, Cornell University, Ithaca, New York

Johnson, William Summer, 1952 (5), Department of Chemistry, University of Wisconsin, Madison 6, Wisconsin

Jones, Donald **F**orsha, 1939 (7), Box 1106, Department of Genetics, Connecticut Agricultural Experiment Station, New Haven 4, Connecticut

Joy, Alfred Harrison, 1944 (2), Mount Wilson Observatory, Pasadena 4, California

Kasner, Edward, 1917 (1), 430 West 116th Street, New York 27, New York

Kaufmann, Berwind Petersen, 1952 (7), Department of Genetics, Carnegie Institution of Washington, Cold Spring Harbor, Long Island, New York

Kelley, Walter Pearson, 1943 (6), 120 Hilgard Hall, University of California, Berkeley 4, California

Kellogg, Arthur Remington, 1951 (8), U. S. National Museum, Smithsonian Institution, Washington 25, D. C.

Kelly, Mervin J., 1945 (4), Bell Telephone Laboratories, 463 West Street, New York 14, New York

Kemble, Edwin Crawford, 1931 (3), Physics Laboratories, Harvard University, Cambridge 38, Mass.

Kendall, Edward Calvin, 1950 (9), 3 Queenston Place, Princeton, New Jersey

Kent, Robert Harrington, 1951 (3), Ballistic Research Laboratories, Ordnance Department, Aberdeen Proving Ground, Maryland

Kerst, Donald William, 1951 (3), Physics Research Laboratory, University of Illinois, Champaign, Illinois

Kettering, Charles Franklin, 1928 (4), General Motors Corporation, Detroit 2, Michigan

Keyes, Frederick George, 1930 (5), Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Kharasch, Morris Selig, 1946 (5), Department of Chemistry, University of Chicago, Chicago 37, Illinois

Kidder, Alfred Vincent, 1936 (11), 10 Frisbie Place, Cambridge 38, Massachusetts

King, Arthur Scott, 1941 (2), Mount Wilson Observatory, Pasadena 4, California

King, Charles Glen, 1951 (9), The Nutrition Foundation, Incorporated, Chrysler Building, New York 17, New York

Kirkwood, John Gamble, 1942 (5), Department of Chemistry, Yale University, New Haven 11, Connecticut

Kistiakowsky, George Bogdan, 1939 (5), Department of Chemistry, 12 Oxford Street, Harvard University, Cambridge 38, Massachusetts

Kluckhohn, Clyde Kay Maben, 1952 (11), Russian Research Center, Harvard University, Cambridge 38, Massachusetts

Knopf, Adolph, 1931 (6), Department of Geology, Stanford University, Stanford, California

Köhler, Wolfgang, 1947 (12), Swarthmore College, Swarthmore, Pennsylvania

Kraus, Charles August, 1925 (5), Brown University, Providence 12, Rhode Island

Kroeber, Alfred L., 1928 (11), Department of Anthropology, Columbia University, New York 27, New York

Kuiper, Gerard Peter, 1950 (2), Yerkes Observatory, University of Chicago, Williams Bay, Wisconsin

Kunkel, Louis Otto, 1932 (7), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Lambert, Walter Davis, 1949 (13), P. O. Box 687, Canaan, Connecticut

LaMer, Victor Kuhn, 1945 (5), Department of Chemistry, Columbia University, New York 27, New York

Langmuir, Irving, 1918 (5), General Electric Company, Schenectady 5, New York

Larsen, Esper S., Jr., 1944 (6), Apt. 502 H, 3930 Connecticut Avenue, N. W., Washington 8, D. C.

Lashley, Karl Spencer, 1930 (12), Yerkes Laboratories of Primate Biology, Orange Park, Florida

Latimer, Wendell Mitchell, 1940 (5), University of California, Berkeley 4, California

Lauritsen, Charles Christian, 1941 (3), California Institute of Technology, Pasadena 4, California

Lawrence, Ernest Orlando, 1934 (3), Radiation Laboratory, University of California, Berkeley 4, California

Lefschetz, Solomon, 1925 (1), Fine Hall, 129 Broadmead Street, Princeton, New Jersey

Leith, Charles Kenneth, 1920 (6), Wardman Park Hotel, Washington 8, D. C.

Leuschner, Armin Otto, 1913 (2), 1816 Scenic Avenue, Berkeley 9, California

Lewis, Howard Bishop, 1949 (9), Department of Biological Chemistry, Medical School, University of Michigan, Ann Arbor, Michigan

Lewis, Warren Harmon, 1936 (8), The Wistar Institute of Anatomy and Biology, Philadelphia 4, Pennsylvania

Lewis, Warren Kendall, 1938 (4), Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Libby, Willard Frank, 1950 (5), Institute for Nuclear Studies, University of Chicago, Chicago 37, Illinois

Lind, Samuel Colville, 1930 (5), P. O. Box P, Oak Ridge, Tennessee

Lindsley, Donald Benjamin, 1952 (12), Department of Psychology, University of California, Los Angeles 24, California

Link, Karl Paul, 1946 (9), Department of Biochemistry, Agricultural Experiment Station, University of Wisconsin, Madison, Wisconsin

Linton, Ralph, 1945 (11), Institute of Human Relations, 333 Cedar Street, New Haven 11, Connecticut

Lipmann, Fritz Albert, 1950 (9), Biochemical Research Laboratory, Massachusetts General Hospital, Boston 14, Massachusetts

Little, Clarence Cook, 1945 (10), Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine

Loeb, Leo, 1937 (10), 40 Crestwood Drive, St. Louis 5, Missouri

Loeb, Robert Frederick, 1946 (9), College of Physicians and Surgeons, 620 West 168th Street, New York 32, New York

Long, Cyril Norman Hugh, 1948 (9), Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut

Long, Esmond Ray, 1946 (10), Henry Phipps Institute, 7th and Lombard Streets, Philadelphia 47, Pennsylvania

Longcope, Warfield Theobald, 1943 (10), Cornhill Farm, Lee, Massachusetts

Longsworth, Lewis Gibson, 1947 (5), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Longwell, Chester Ray, 1935 (6), Kirtland Hall, Yale University, New Haven, Connecticut

Loomis, Alfred Lee, 1941 (4), The Loomis Laboratory, Room 2420, 14 Wall St., New York 5, New York

Loomis, Francis Wheeler, 1949 (3), Massachusetts Institute of Technology, P. O. Box 390, Cambridge 39, Massachusetts

Lorente de Nó, Rafael, 1950 (9), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Lothrop, Samuel Kirkland, 1951 (11), Peabody Museum, Harvard University, Cambridge 38, Massachusetts

Lovering, Thomas Seward, 1949 (6), 8001 West 17th Avenue, Lakewood, Colorado

Lowie, Robert Harry, 1931 (11), University of California, Berkeley 4, California

Lyman, Theodore, 1917 (3), Research Laboratory of Physics, Harvard University, Cambridge 38, Massachusetts

McClintock, Barbara, 1944 (7), Carnegie Institution, Cold Spring Harbor, Long Island, New York

McCollum, Elmer Verner, 1920 (9), Gilman Hall, Johns Hopkins University, Baltimore 18, Maryland

McElvain, Samuel Marion, 1949 (5), Department of Chemistry, University of Wisconsin, Madison 6, Wisconsin

McMaster, Philip Duryeé, 1952 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

McMillan, Edwin Mattison, 1947 (3), Radiation Laboratory, University of California, Berkeley 4, California

McShane, Edward James, 1948 (1), School of Mathematics, University of Virginia, Charlottesville, Virginia

MacInnes, Duncan Arthur, 1937 (5), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Mac Lane, Saunders, 1949 (1), Department of Mathematics, University of Chicago, Chicago 37, Illinois

Macelwane, James Bernard, S.J., 1944 (13), 221 North Grand Boulevard, St. Louis 3, Missouri

Mangelsdorf, Paul Christoph, 1945 (7), Botanical Museum, Harvard University, Cambridge 38, Massachusetts

Mann, Frank Charles, 1950 (9), Mayo Foundation, University of Minnesota, Rochester, Minnesota

Marshall, Eli Kennerly, Jr., 1943 (9), School of Medicine, Johns Hopkins University, 710 North Washington Street, Baltimore 5, Maryland

Marvel, Carl Shipp, 1938 (5), Department of Chemistry, University of Illinois, Urbana, Illinois

Mason, Max, 1923 (3), 1035 Harvard Avenue, Claremont, California

Maxcy, Kenneth Fuller, 1950 (10), School of Hygiene and Public Health, Johns Hopkins University, 615 North Wolfe Street, Baltimore 5, Maryland

Mayall, Nicholas Ulrich, 1949 (2), University of California, Lick Observatory, Mount Hamilton, California

Mayer, Joseph Edward, 1946 (5), Department of Chemistry, University of Chicago, Chicago 37, Illinois

Maynard, Leonard Amby, 1944 (9), School of Nutrition, Cornell University, Ithaca, New York

Mead, Warren Judson, 1939 (6), Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Meek, Walter Joseph, 1947 (9), Department of Physiology, University of Wisconsin, Madison 6, Wisconsin

Mees, Charles Edward Kenneth, 1950 (5), Eastman Kodak Company, Kodak Park Works, Rochester 4, New York

Mendenhall, Walter Curran, 1932 (6), 9 East Lenox Street, Chevy Chase 15, Maryland

Menzel, Donald Howard, 1948 (2), Harvard College Observatory, Cambridge 38, Massachusetts

Merica, Paul Dyer, 1942 (4), 67 Wall Street, New York 5, New York

Merrill, Paul Willard, 1929 (2), Mount Wilson Observatory, Pasadena 4, California

Metz, Charles William, 1948 (8), University of Pennsylvania, Zoological Laboratory, 38th Street and Woodland Avenue, Philadelphia 4, Pennsylvania

Meyer, Karl Friederich, 1940 (10), George Williams Hooper Foundation, University of California Medical Center, San Francisco 22, California

Miles, Walter Richard, 1933 (12), Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut

Millikan, Robert Andrews, 1915 (3), California Institute of Technology, Pasadena 4, California

Mitchell, Samuel Alfred, 1933 (2), Leander McCormick Observatory, University Station, Charlottesville, Virginia

Moore, Carl Richard, 1944 (8), University of Chicago, Chicago 37, Illinois

Moore, Robert Lee, 1931 (1), University of Texas, Austin 12, Texas

Morse, Harold Marston, 1932 (1), The Institute for Advanced Study, Princeton, New Jersey

Mueller, John Howard, 1945 (10), Harvard Medical School, 25 Shattuck Street, Boston 15, Massachusetts

Muller, Hermann Joseph, 1931 (8), Science Hall 101, Indiana University, Bloomington, Indiana

Mulliken, Robert Sanderson, 1936 (3), University of Chicago, Chicago 37, Illinois

Murnaghan, Francis Dominic, 1942 (1), Instituto Tecnologico de Aeronautica, São Jose dos Campos, Estado de São Paulo, Brasil

Murphree, Eger Vaughan, 1950 (4), Standard Oil Development Company, 15 West 51st Street, New York 19, New York

Nicholas, John Spangler, 1949 (8), Osborn Zoological Laboratory, Yale University, New Haven 11, Connecticut

Nicholson, Seth Barnes, 1937 (2), Mount Wilson Observatory, Pasadena 4, California

Niemann, Carl George, 1952 (5), Gates and Crellin Laboratories of Chemistry, California Institute of Technology, Pasadena 4, California

Nier, Alfred Otto C., 1950 (3), Department of Physics, University of Minnesota, Minneapolis 14, Minnesota

Nolan, Thomas Brennan, 1951 (6), Geological Survey, U. S. Department of the Interior, Washington 25, D. C.

Northrop, John Howard, 1934 (9), Department of Bacteriology, University of California, Berkeley 4, California

Novy, Frederick George, 1924 (10), 721 Forest Avenue, Ann Arbor, Michigan

Noyes, William Albert, Jr., 1943 (5), Department of Chemistry, University of Rochester, Rochester 3, New York

Oncley, John Lawrence, 1947 (9), University Laboratory of Physical Chemistry, Harvard University, 25 Shattuck St., Boston 15, Massachusetts

Ons ger, Lars, 1947 (5), Sterling Chemistry Laboratory, Yale University, 225 Prospect Street, New Haven, Connecticut

Opie, Eugene Lindsay, 1923 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Oppenheimer, J. Robert, 1941 (3), The Institute for Advanced Study, Princeton, New Jersey

Osterhout, Winthrop John Vanleuven, 1919 (7), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Painter, Theophilus Shickel, 1938 (8), University of Texas, Austin 12, Texas

Palache, Charles, 1934 (6), Harvard University, Cambridge 38, Massachusetts

Patterson, John Thomas, 1941 (8), Department of Zoology, University of Texas, Austin 12, Texas

Paul, John Rodman, 1945 (10), Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut

Pauling, Linus, 1933 (5), Crellin Laboratory, California Institute of Technology, Pasadena 4, California

Pegram, George Braxton, 1949 (3), Columbia University, New York 27, New York

Pekeris, Chaim Leib, 1952 (13), Department of Applied Mathematics, The Weizmann Institute of Science, Rehovoth, Israel

Peters, John Punnett, 1947 (9), Department of Internal Medicine, Yale University School of Medicine, New Haven 11, Connecticut

Pierce, George Washington, 1920 (3), Cruft Laboratory, Harvard University, Cambridge 38, Massachusetts

Piggot, Charles Snowden, 1946 (13), The Cosmos Club, Washington 5, D. C.

Pillsbury, Walter Bowers, 1925 (12), University of Michigan, Ann Arbor, Michigan

Pitzer, Kenneth Sanborn, 1949 (5), Gilman Hall, University of California, Berkeley 4, California

Purcell, Edward Mills, 1951 (3), Lyman Laboratory of Physics, Harvard University, Cambridge 38, Massachusetts

Rabi, Isidor Isaac, 1940 (3), Department of Physics, Columbia University, New York 27, New York

Ramsey, Norman Foster, 1952 (3), Lyman Laboratory of Physics, Harvard University, Cambridge 38, Massachusetts

Raper, Kenneth Bryan, 1949 (7), Northern Regional Research Laboratory, U. S. Department of Agriculture, Peoria 5, Illinois

Raymond, Arthur Emmons, 1950 (4), Douglas Aircraft Company, Incorporated, 3000 Ocean Park Boulevard, Santa Monica, California

Reeside, John Bernard, Jr., 1945 (6), Box 177, Hyattsville, Maryland

Reichelderfer, Francis Wilton, 1945 (13), Room 310, U. S. Weather Bureau, Washington 25, D. C.

Rhoades, Marcus Morton, 1946 (7), Department of Botany, University of Illinois, Urbana, Illinois

Richards, Alfred Newton, 1927 (9), School of Medicine, University of Pennsylvania, Philadelphia 4, Pennsylvania

Richter, Curt Paul, 1948 (12), Johns Hopkins Hospital, Baltimore 5, Maryland

Riddle, Oscar, 1939 (8), Route 4, Plant City, Florida

Riker, Albert Joyce, 1951 (7), Department of Plant Pathology, University of Wisconsin, Madison 6, Wisconsin

Rivers, Thomas Milton, 1934 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Robbins, William Jacob, 1940 (7), New York Botanical Garden, Bronx Park (Fordham Station), New York 58, New York

Robertson, Howard Percy, 1951 (3), Weapons Systems Evaluation Group, Office of the Secretary of Defense, Washington 25, D. C.

Robertson, Oswald Hope, 1943 (10), Department of Biology, Stanford University, Stanford, California

Rodebush, Worth Huff, 1938 (5), University of Illinois, Urbana, Illinois

Romer, Alfred Sherwood, 1944 (8), Museum of Comparative Zoology, Oxford Street, Cambridge 38, Massachusetts

Rose, William Cumming, 1936 (9), University of Illinois, Urbana, Illinois

Ross, Frank Elmore, 1930 (2), Mount Wilson Observatory, Pasadena 4, California

Rossby, Carl-Gustaf Arvid, 1943 (13), Institute of Meteorology, University of Chicago, Chicago 37, Illinois

Rossi, Bruno Benedetto, 1950 (3), Department of Physics, Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Rossini, Frederick Dominic, 1951 (5), Department of Chemistry, Carnegie Institute of Technology, Pittsburgh 13, Pennsylvania

Rous, Francis **Peyton,** 1927 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Rubey, William Walden, 1945 (6), Geological Survey, U. S. Department of the Interior, Washington 25, D. C.

Ruedemann, Rudolf, 1928 (6), 161 Dana Avenue, Albany 3, New York

Russell, Henry Norris, 1918 (2), Princeton University, Princeton, New Jersey

Sabin, Albert Bruce, 1951 (10), The Children's Hospital Research Foundation, Elland Avenue and Bethesda, Cincinnati 29, Ohio

Sabin, Florence Rena, 1925 (10), 1333 East 10th Avenue, Denver 3, Colorado

Saunders, Frederick Albert, 1925 (3), South Hadley, Massachusetts

Savage, John **Lucian,** 1949 (4), 1651 Dahlia Street, Denver 7, Colorado

Sax, Karl, 1941 (7), Harvard University, Cambridge 38, Massachusetts

Scatchard, George, 1946 (5), Department of Chemistry, Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Schlesinger, Hermann Irving, 1948 (5), Department of Chemistry, University of Chicago, Chicago 37, Illinois

Schmidt, Carl Frederic, 1949 (9), Laboratory of Pharmacology, School of Medicine, University of Pennsylvania, Philadelphia 4, Pennsylvania

Schmitt, Francis Otto, 1948 (8), Department of Biology, Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Schrader, Franz, 1951 (8), Department of Zoology, Columbia University, New York 27, New York

Schultz, Adolph Hans, 1939 (11), Anthropologisches Institut, Sempersteig 3, Zurich, Switzerland

Schwinger, Julian, 1949 (3), Department of Physics, Harvard University, Cambridge 38, Massachusetts

Seaborg, Glenn Theodore, 1948 (5), Department of Chemistry, University of California, Berkeley 4, California

Seares, Frederick **Hanley,** 1919 (2), 351 Palmetto Drive, Pasadena 2, California

Segrè, Emilio, 1952 (3), Department of Physics, University of California, Berkeley 4, California

Seitz, Frederick, 1951 (3), Department of Physics, University of Illinois, Urbana, Illinois

Serber, Robert, 1952 (3), Department of Physics, Columbia University, New York 27, New York

Shaffer, Philip Anderson, 1928 (9), Washington University Medical School, St. Louis 10, Missouri

Shapiro, Harry **Lionel,** 1949 (11), Department of Anthropology, American Museum of Natural History, Central Park West at 79th Street, New York 24, New York

Shapley, Harlow, 1924 (2), Harvard College Observatory, Cambridge 38, Massachusetts

Sherman, Henry **Clapp,** 1933 (9), Columbia University, New York 27, New York

Shockley, William, 1951 (4), Bell Telephone Laboratories, Murray Hill, New Jersey

Shope, Richard Edwin, 1940 (10), Box 323, Kingston, New Jersey

Simpson, George Gaylord, 1941 (6), American Museum of Natural History, 77th Street and Central Park West, New York 24, New York

Sinnott, Edmund Ware, 1936 (7), Hall of Graduate Studies, Yale University, New Haven, Connecticut

Skinner, Burrhus Frederic, 1950 (12), Psychological Laboratories, Harvard University, Cambridge 38, Massachusetts

Slater, John Clarke, 1932 (3), Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Slepian, Joseph, 1941 (4), Westinghouse Electric Corporation, East Pittsburgh, Pennsylvania

Slichter, Louis Byrne, 1944 (13), 1446 North Amalfi Drive, Pacific Palisades, California

Slipher, Vesto Melvin, 1921 (2), Lowell Observatory, Flagstaff, Arizona

Small, Lyndon Frederick, 1941 (5), National Institutes of Health, Bethesda 14, Maryland

Smith, Gilbert Morgan, 1948 (7), School of Biological Sciences, Stanford University, Stanford, California

Smith, Homer William, 1945 (9), 477 First Avenue, New York 16, New York

Smith, Lee Irvin, 1944 (5), School of Chemistry, University of Minnesota, Minneapolis 14, Minnesota

Smith, Paul Althaus, 1947 (1), Department of Mathematics, Columbia University, New York 27, New York

Smith, Philip Edward, 1939 (8), College of Physicians and Surgeons, 630 West 168th Street, New York 32, New York

Soderberg, Carl Richard, 1947 (4), Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Sonneborn, Tracy Morton, 1946 (8), Department of Zoology, Indiana University, Bloomington, Indiana

Spedding, Frank Harold, 1952 (5), Institute for Atomic Research, Iowa State College, Ames, Iowa

Spier, Leslie, 1946 (11), P. O. Box 880, Santa Cruz, California

Spitzer, Lyman, Jr., 1952 (2), Princeton University Observatory, 14 Prospect Avenue, Princeton, New Jersey

Stadie, William Christopher, 1945 (9), 821 Maloney Clinic, 36th and Spruce Streets, Philadelphia 4, Pennsylvania

Stadler, Lewis John, 1938 (7), University of Missouri, Columbia, Missouri

Stakman, Elvin Charles, 1934 (7), University Farm, St. Paul 8, Minnesota

Stanley, Wendell Meredith, 1941 (9), Department of Biochemistry, University of California, Berkeley 4, California

Stebbins, George Ledyard, Jr., 1952 (7), Division of Genetics, University of California, Agricultural Experiment Station, Davis, California

Stebbins, Joel, 1920 (2), Lick Observatory, Mt. Hamilton, California

Stern, Curt, 1948 (8), Department of Zoology, University of California, Berkeley 4, California

Stern, Otto, 1945 (3), 759 Cragmont Avenue, Berkeley, California

Stevens, Stanley Smith, 1946 (12), Department of Psychology, Harvard University, Cambridge 38, Massachusetts

Stewart, George Walter, 1938 (3), State University of Iowa, Iowa City, Iowa

Stone, Calvin Perry, 1943 (12), Stanford University, Stanford, California

Stone, Marshall Harvey, 1938 (1), 313 Eckhart Hall, University of Chicago, Chicago 37, Illinois

Stratton, Julius Adams, 1950 (4), Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Struve, Otto, 1937 (2), Department of Astronomy, University of California, Berkeley 4, California

Sturtevant, Alfred Henry, 1930 (8), California Institute of Technology, Pasadena 4, California

Suits, Chauncey Guy, 1946 (4), Research Laboratory, General Electric Company, Schenectady 5, New York

Sumner, James Batcheller, 1948 (9), New York State College of Agriculture, Laboratory of Enzyme Chemistry, Cornell University, Ithaca, New York

Taliaferro, William Hay, 1940 (8), University of Chicago, Chicago 37, Illinois

Tatum, Edward Lawrie, 1952 (9), Division of Biological Sciences, Stanford University, Stanford, California

Teller, Edward, 1948 (3), Institute for Nuclear Studies, University of Chicago, Chicago 37, Illinois

Terman, Frederick Emmons, 1946 (4), School of Engineering, Stanford University, Stanford, California

Terman, Lewis Madison, 1928 (12), 761 Dolores Street, Stanford, California

Thimann, Kenneth Vivian, 1948 (7), Harvard University, Biological Laboratories, 16 Divinity Avenue, Cambridge 38, Massachusetts

Thom, Charles, 1937 (7), 207 Grant Street, Port Jefferson, New York

Thomas, Charles Allen, 1948 (4), Monsanto Chemical Company, 1700 South Second Street, St. Louis 4, Missouri

Thomas, Tracy Yerkes, 1941 (1), Swain Hall, Indiana University, Bloomington, Indiana

Thompson, Thomas Gordon, 1951 (13), Oceanographic Laboratories, University of Washington, Seattle 5, Washington

Thurstone, Louis Leon, 1938 (12), University of Chicago, Chicago 37, Illinois

Tillett, William Smith, 1951 (10), New York University College of Medicine, 477 First Avenue, New York 16, New York

Timoshenko, Stephen Prokop, 1940 (4), Room 262, Engineering Building, Stanford University, Stanford, California

Tolman, Edward Chace, 1937 (12), Department of Psychology, University of California, Berkeley 4, California

Tozzer, Alfred Marston, 1942 (11), Peabody Museum, Harvard University, Cambridge, 38, Massachusetts

Trumpler, Robert Julius, 1932 (2), Route 2, Box 1750, Aptos, California

Tuve, Merle Antony, 1946 (3), Department of Terrestrial Magnetism, Carnegie Institution of Washington, 5241 Broad Branch Road, N. W., Washington 15, D. C.

Twitty, Victor Chandler, 1950 (8), Department of Biological Sciences, Stanford University, Stanford, California

Tyzzer, Ernest Edward, 1942 (10), 175 Water Street, Wakefield, Massachusetts

Urey, Harold Clayton, 1935 (5), Institute for Nuclear Studies, University of Chicago, Chicago 37, Illinois

Vandiver, Harry Shultz, 1934 (1), Department of Applied Mathematics, University of Texas, Austin 12, Texas

Van Niel, Cornelis Bernardus, 1945 (7), Hopkins Marine Station of Stanford University, Pacific Grove, California

Van Slyke, Donald Dexter, 1921 (9), Brookhaven National Laboratory, Upton, Long Island, New York

Van Vleck, John Hasbrouck, 1935 (3), Harvard University, Cambridge 38, Massachusetts

Veblen, Oswald, 1919 (1), The Institute for Advanced Study, Princeton. New Jersey

Vickery, Hubert Bradford, 1943 (9), Connecticut Agricultural Experiment Station, New Haven 4, Connecticut

von Kármán, Theodore, 1938 (4), 1501 South Marengo Avenue, Pasadena, California

von Neumann, John, 1937 (1), The Institute for Advanced Study, Princeton, New Jersey

Waksman, Selman Abraham, 1942 (7), Agricultural Experiment Station, New Brunswick, New Jersey

Wald, George, 1950 (9), Biological Laboratories, Harvard University, 16 Divinity Avenue, Cambridge 38, Massachusetts

Walker, John Charles, 1945 (7), 206 Horticulture Building, University of Wisconsin, Madison 6, Wisconsin

Walsh, Joseph Leonard, 1936 (1), Harvard University, Cambridge 38, Massachusetts

Webster, David Locke, 1923 (3), Physics Department, Room 385, Stanford University, Stanford, California

Weiss, Paul Alfred, 1947 (8), Department of Zoology, University of Chicago, Chicago 37, Illinois

Weisskopf, Victor Frederick, 1952 (3), Department of Physics, Massachusetts Institute of Technology, Cambridge 39, Massachusetts

Went, Frits Warmolt, 1947 (7), California Institute of Technology, Pasadena 4, California

Werkman, Chester Hamlin, 1946 (9), Department of Bacteriology, Iowa State College, Ames, Iowa

Wetmore, Alexander, 1945 (8), Smithsonian Institution, Washington 25, D. C.

Wever, Ernest Glen, 1940 (12), Princeton University, Princeton, New Jersey

Weyl, Claus Hugo Hermann, 1940 (1), Bergstrasse 27, Zurich, Switzerland

Wheeler, John Archibald, 1952 (3), Palmer Physical Laboratory, Princeton University, Princeton, New Jersey

Whipple, George Hoyt, 1929 (10), School of Medicine and Dentistry, University of Rochester, 260 Crittenden Boulevard, Rochester 7, New York

Whitehead, John Boswell, 1932 (4), Johns Hopkins University, Baltimore 18, Maryland

Whitney, Hassler, 1945 (1), Department of Mathematics, Harvard University, Cambridge 38, Massachusetts

Whyburn, Gordon Thomas, 1951 (1), School of Mathematics, University of Virginia, Charlottesville, Virginia

Wiggers, Carl John, 1951 (9), School of Medicine, Western Reserve University, Cleveland 6, Ohio

Wigner, Eugene Paul, 1945 (3), 8 Ober Road, Princeton, New Jersey

Williams, Howel, 1950 (6), Department of Geological Sciences, University of California, Berkeley 4, California

Williams, John Warren, 1952 (5), Department of Chemistry, University of Wisconsin, Madison 6, Wisconsin

Williams, Robert R., 1945 (5), 297 Summit Avenue, Summit, New Jersey

Williams, Roger John, 1946 (5), Biochemical Institute, University of Texas, Austin 12, Texas

Willier, Benjamin Harrison, 1945 (8), Department of Biology, Johns Hopkins University, Baltimore 18, Maryland

Wilson, Edgar Bright, Jr., 1947 (5), Harvard University, Department of Chemistry, 12 Oxford Street, Cambridge 38, Massachusetts

Wilson, Edwin Bidwell, 1919 (3), Harvard School of Public Health, 695 Huntington Avenue, Boston 15, Massachusetts

Wilson, Ralph Elmer, 1950 (2), 605 Acacia Avenue, Corona del Mar, California

Wilson, Robert Erastus, 1947 (4), 910 South Michigan Avenue, Chicago 80, Illinois

Wintersteiner, Oskar, 1950 (9), Squibb Institute for Medical Research, New Brunswick, New Jersey

Wislocki, George Bernays, 1941 (8), Harvard Medical School, 25 Shattuck Street, Boston 15, Massachusetts

Wolbach, Simeon Burt, 1938 (10), Department of Pathology, Children's Hospital, 300 Longwood Avenue, Boston 15, Massachusetts

Wolfrom, Melville Lawrence, 1950 (5), Department of Chemistry, Ohio State University, Columbus 10, Ohio

Wood, Robert Williams, 1912 (3), Johns Hopkins University, Baltimore 18, Maryland

Woodring, Wendell Phillips, 1946 (6), Geological Survey, U. S. Department of the Interior, Washington 25, D. C.

Woodworth, Robert Sessions, 1921 (12), Columbia University, New York 27, New York

Woolley, Dilworth Wayne, 1952 (9), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, New York

Wright, Frederick Eugene, 1923 (6), 2134 Wyoming Avenue, N. W., Washington 8, D. C.

Wright, Sewall Green, 1934 (8), Department of Zoology, University of Chicago, Chicago 37, Illinois

Wright, William Hammond, 1922 (2), 60 North Keeble Avenue, San Jose 26, California

Wulf, Oliver Reynolds, 1949 (13), Weather Bureau Regional Office, Gates and Crellin Laboratories, California Institute of Technology, Pasadena 4, California

Wyckoff, Ralph Walter Graystone, 1949 (5), Laboratory of Physical Biology, National Institutes of Health, Bethesda 14, Maryland

Yerkes, Robert Mearns, 1923 (12), Yale University School of Medicine, 333 Cedar Street, New Haven, Connecticut

Yost, Don Merlin Lee, 1944 (5), California Institute of Technology, Pasadena 4, California

Young, William Gould, 1951 (5), Department of Chemistry, University of California, Los Angeles 24, California

Zachariasen, Frederik **William** Houlder, 1949 (3), Department of Physics, University of Chicago, Chicago 37, Illinois

Zariski, Oscar, 1944 (1), Department of Mathematics, Harvard University, Cambridge 38, Massachusetts

Zworykin, Vladimir Kosma, 1943 (4), 103 Battle Road, Princeton, New Jersey

Number of Members July 1, 1952: 499.

MEMBERS EMERITI

Allen, Charles Elmer, 1924, Biology Building, University of Wisconsin, Madison 6, Wisconsin

Anderson, John August, 1928, P. O. Box 332, Pasadena 17, California

Benedict, Francis Gano, 1914, Machiasport, Maine

Campbell, Douglas Houghton, 1910, Stanford University, Stanford, California

Child, Charles Manning, 1935, Jordan Hall, Stanford University, Stanford, California

Dickson, Leonard Eugene, 1913, Route #2, Joliet, Illinois
Hulett, George Augustus, 1922, 44 Washington Road, Princeton, New Jersey
Merrill, Elmer Drew, 1923, Arnold Arboretum, Jamaica Plain, Massachusetts
Moulton, Forest Ray, 1910, 1637 Orrington Street, Evanston, Illinois
Parker, George Howard, 1913, Harvard Biological Laboratories, Divinity Avenue, Cambridge 38, Massachusetts
Stratton, George Malcolm, 1928, University of California, Berkeley 4, California
Swanton, John Reed, 1932, 22 George Street, Newton 58, Massachusetts
Whitney, Willis Rodney, 1917, General Electric Company, Schenectady 5, New York

FOREIGN ASSOCIATES

The number in parentheses following the year of election indicates association within the sections of the National Academy of Sciences.

Adrian, Edgar Douglas, 1941 (9), Trinity College, Cambridge, England
Alexandroff, Paul A., 1947 (1), Mathematical Institute of the Academy of Sciences of the U.S.S.R., Bolshaya Kalushskaya 19, Moscow, U.S.S.R.
Bailey, Sir Edward, 1944 (6), 19 Greenhill Gardens, Edinburgh 10, Scotland
Bartlett, Sir Frederic Charles, 1947 (12), University of Cambridge, The Psychological Laboratory, Downing Place, Cambridge, England
Best, Charles Herbert, 1950 (9), Banting and Best Department of Medical Research, University of Toronto, Toronto, Canada
Bjerrum, Niels, 1952 (5), The Royal Veterinary and Agricultural College, Copenhagen, Denmark
Bohr, Niels, 1925 (3), Institute for Theoretical Physics, Blegdamsvej 15, Copenhagen, Denmark
Bordet, Jules, 1935 (9), Pasteur Institute, Rue du Remorquer, 28, Brussels, Belgium
Bragg, Sir William Lawrence, 1945 (3), Cavendish Laboratory, The University, Cambridge, England
de Broglie, Prince Louis, 1948 (3), 94 Rue Perronet, Neuilly-sur-Seine, France
Caso, Alfonso, 1943 (11), Av. Sudderman No. 312, Col. Chapultepec-Morales, Mexico 5, D. F.
Chapman, Sydney, 1946 (1), Letters: Queen's College, Oxford; Printed matter: The Mathematical Institute, Oxford, England
Dale, Sir Henry Hallett, 1940 (9), The Wellcome Trust, 28 Portman Square, London, W.1, England

Dirac, Paul Adrien Maurice, 1949 (1), Department of Mathematics, St. John's College, Cambridge, England

Eskola, Pentti Eelis, 1951 (6), Helsinki University, Snellmanink. 5, Helsinki, Finland

Fisher, Ronald Aylmer, 1948 (8), Department of Genetics, University of Cambridge, 44 Storey's Way, Cambridge, England

von Frisch, Karl, 1951 (8), The Zoological Institute, University of Munich, Munich, Germany

Hadamard, Jacques, 1926 (1), 12 Rue Emile Faguet, Paris XIV, France

Helland-Hansen, Björn, 1947 (13), Chr. Michelsens Institutt for Videnscap, Bergen, Norway

Hill, Archibald Vivian, 1941 (9), 16 Bishopswood Road, Highgate, London, N.6, England

Hill, James Peter, 1940 (8), Kanimbla, Dollis Avenue, London N.3, England

Houssay, Bernardo Alberto, 1940 (9), Viamonte 2790, Buenos Aires, Argentina

Jeffreys, Harold, 1945 (13), St. John's College, Cambridge, England

Jones, Sir Harold Spencer, 1943 (2), Royal Greenwich Observatory, Herstmonceux Castle, Hailsham, Sussex, England

Kapitza, Peter Leonidovich, 1946 (3), Institute for Physical Problems, Academy of Sciences of the U.S.S.R., Moscow, U.S.S.R.

Karrer, Paul, 1945 (5), University of Zurich, Zurich, Switzerland

Keith, Sir Arthur, 1941 (11), Buckston Browne Farm, Downe, Farnborough, Kent, England

Kluyver, Albert Jan, 1950 (7), Technical University, Delft, The Netherlands

Levi, Giuseppe, 1940 (8), Instituto di Anatomia Umana, Corso Massimo D'Azeglio, 52, Turin, Italy

Lim, Robert K. S., 1942 (9), The Miles-Ames Research Laboratory, Elkhart, Indiana

Linderstrøm-Lang, Kaj Ulrik, 1947 (5), Chemical Division, Carlsberg Laboratory, Copenhagen, Denmark

Pieron, Henri, 1949 (12), Institute of Psychology, University of Paris, Paris, France

Reichstein, Tadeus, 1952 (5), Organic Laboratories, University of Basel, Basel, Switzerland

Robinson, Sir Robert, 1934 (5), Dyson Perrins Laboratory, South Parks Road, Oxford, England

Ruzicka, Leopold, 1944 (5), Department of Organic Chemistry, Institute of Technology, Zurich, Switzerland

Southwell, Richard Vynne, 1943 (4), Imperial College of Science and Technology, South Kensington, London, S.W.7, England

Svedberg, The, 1945 (5), Fysikalisk-Kemiska Institutionen, University of Uppsala, Uppsala, Sweden

Sverdrup, Harald Ulrik, (1945)* 1952 (13), Norsk Polarinstitutt, Observatoriegt 1, Oslo, Norway

Taylor, Sir Geoffrey I., 1945 (1), Trinity College, Cambridge, England

Thomson, Sir Godfrey, 1951 (12), Edinburgh University, 5 Ravelston Dykes, Edinburgh 4, Scotland

Tiselius, Arne W. K., 1949 (9), Institute of Biochemistry, Uppsala University, Uppsala, Sweden

Vallee-Poussin, C. de la, 1929 (1), University of Louvain, Louvain, Belgium

Vening Meinesz, Felix Andries, 1939 (13), Potgieterlaan 5, Amersfoort, The Netherlands

Watson, D. M. S., 1938 (8), University College, Gower Street, London, W.C.1, England; Calendar year 1952, Museum of Comparative Zoology, Harvard College, Cambridge 38, Massachusetts

Wieland, Heinrich, 1932 (5), Sophienstrasse 9, Munich 2 NW, Germany

Winge, Öjvind, 1949 (9), Department of Physiology, Carlsberg Laboratory, Copenhagen (Valby), Denmark

Yukawa, Hideki, 1949 (3), Department of Physics, Columbia University, New York 27, New York

* Dr. Sverdrup was elected a member of the Academy in 1945. He resigned on April 2, 1951, on which date he had maintained residence in Norway for three years, thereby losing his American citizenship. He was elected a foreign associate on April 29, 1952.

Number of Foreign Associates July 1, 1952: 47

SECTIONS

(1) *Mathematics*—27 members

McShane, E. J., *Chairman* (1955)
Albert, A. A.
Alexander, J. W.
Bell, E. T.
Bochner, S.
Chevalley, C.
Coble, A. B.
Douglas, Jesse
Eisenhart, L. P.
Evans, G. C.
Kasner, Edward
Lefschetz, Solomon
Mac Lane, Saunders
Moore, R. L.
Morse, Marston
Murnaghan, F. D.
Smith, Paul A.
Stone, M. H.
Thomas, T. Y.
Vandiver, H. S.
Veblen, Oswald
von Neumann, John
Walsh, J. L.
Weyl, Hermann
Whitney, Hassler
Whyburn, G. T.
Zariski, O.

Foreign Associates

Alexandroff, P. A.
Chapman, Sydney
Dirac, P. A. M.
Hadamard, Jacques
Taylor, Sir Geoffrey
Vallee-Poussin, C. de la

(2) *Astronomy*—27 members

Merrill, P. W., *Chairman* (1953)
Abbott, C. G.
Adams, W. S.
Babcock, H. D.
Bowen, I. S.
Brouwer, Dirk
Clemence, G. M.
Hubble, E. P.
Joy, A. H.
King, A. S.
Kuiper, G. P.
Leuschner, A. O.
Mayall, N. U.
Menzel, D. H.
Mitchell, S. A.
Nicholson, S. B.
Ross, F. E.
Russell, H. N.
Seares, F. H.
Shapley, Harlow
Slipher, V. M.
Spitzer, Lyman, Jr.
Stebbins, Joel
Struve, Otto
Trumpler, R. J.
Wilson, Ralph E.
Wright, W. H.

Foreign Associate

Jones, Sir H. Spencer

(3) *Physics*—69 members

Slater, J. C., *Chairman* (1954)
Allison, S. K.
Alvarez, L. W.
Anderson, C. D.
Bacher, R. F.
Bainbridge, K. T.
Beams, J. W.
Bethe, H. A.
Birge, R. T.
Bloch, Felix
Bradbury, N. E.
Breit, Gregory
Bridgman, P. W.
Brode, R. B.
Coblentz, W. W.
Compton, A. H.
Compton, K. T.
Condon, E. U.
Coolidge, W. D.
Crew, Henry
Davis, Bergen
Davisson, C. J.
DuBridge, L. A.
Dunning, J. R.
Einstein, Albert
Epstein, P. S.

Fermi, Enrico
Franck, James
Goudsmit, S. A.
Houston, W. V.
Hull, A. W.
Ives, H. E.
Kemble, E. C.
Kent, R. H.
Kerst, D. W.
Lauritsen, C. C.
Lawrence, E. O.
Loomis, F. W.
Lyman, Theodore
McMillan, E. M.
Mason, Max
Millikan, R. A.
Mulliken, R. S.
Nier, A. O. C.
Oppenheimer, J. R.
Pegram, G. B.
Pierce, G. W.
Purcell, E. M.
Rabi, I. I.
Ramsey, N. F.
Robertson, H. P.
Rossi, Bruno
Saunders, F. A.
Schwinger, Julian
Segrè, E.
Seitz, Frederick
Serber, R.
Stern, Otto
Stewart, G. W.
Teller, Edward
Tuve, M. A.
Van Vleck, J. H.
Webster, D. L.
Weisskopf, V. F.
Wheeler, J. A.
Wigner, E. P.
Wilson, Edwin B.
Wood, R. W.
Zachariasen, W. H.

Foreign Associates

Bohr, Niels
Bragg, Sir Lawrence
de Broglie, Prince Louis
Kapitza, P. L.
Yukawa, Hideki

(4) *Engineering*—38 members

Hunsaker, J. C., *Chairman* (1953)
Adams, C. A.
Briggs, L. J.
Buckley, O. E.
Bush, Vannevar
Cochrane, E. L.
Curme, G. O., Jr.
Dryden, H. L.
Dunn, Gano
Durand, W. F.
Fletcher, Harvey
Foote, P. D.
Gibbs, W. F.
Gilliland, E. R.
Greenewalt, C. H.
Herty, C. H., Jr.
Hoover, Herbert
Jeffries, Zay
Kelly, M. J.
Kettering, C. F.
Lewis, W. K.
Loomis, A. L.
Merica, P. D.
Murphree, E. V.
Raymond, A. E.
Savage, J. L.
Shockley, W.
Slepian, Joseph
Soderberg, C. R.
Stratton, J. A.
Suits, C. G.
Terman, F. E.
Thomas, C. A.
Timoshenko, Stephen
von Kármán, T.
Whitehead, J. B.
Wilson, Robert E.
Zworykin, V. K.

Foreign Associate

Southwell, R. V.

(5) *Chemistry*—68 members

Fuson, R. C., *Chairman* (1953)
Adams, Roger
Badger, R. M.
Bancroft, W. D.
Bartlett, P. D.
Baxter, G. P.
Bogert, M. T.
Bolton, E. K.
Conant, J. B.
Cope, A. C.
Craig, L. C.
Daniels, Farrington
Debye, Peter
Edsall, J. T.
Elderfield, R. C.
Eyring, Henry
Fieser, L. F.
Folkers, Karl
Fuoss, R. M.
Giauque, W. F.
Gilman, Henry
Hammett, L. P
Harned, H. S.

Hendricks, S. B.
Hildebrand, J. H.
Hudson, C. S.
Ipatieff, V. N.
Jacobs, W. A.
Johnson, J. R.
Johnson, W. S.
Keyes, F. G.
Kharasch, M. S.
Kirkwood, J. G.
Kistiakowsky, G. B.
Kraus, C. A.
La Mer, V. K.
Langmuir, Irving
Latimer, W. M.
Libby, W. F.
Lind, S. C.
Longsworth, L. G.
McElvain, S. M.
MacInnes, D. A.
Marvel, C. S.
Mayer, J. E.
Mees, C. E. K.
Niemann, C. G.
Noyes, W. A., Jr.
Onsager, Lars
Pauling, Linus
Pitzer, K. S.
Rodebush, W. H.
Rossini, F. D.
Scatchard, George
Schlesinger, H. I.
Seaborg, G. T.
Small, L. F.
Smith, L. I.
Spedding, F. H.
Urey, H. C.
Williams, J. W.
Williams, Robert R.
Williams, Roger J.
Wilson, E. Bright, Jr.
Wolfrom, M. L.
Wyckoff, R. W.
Yost, D. M.
Young, W. G.

Foreign Associates

Bjerrum, Niels
Karrer, Paul
Linderstrøm-Lang, K. U.
Reichstein, Tadeus
Robinson, Sir Robert
Ruzicka, Leopold
Svedberg, The
Wieland, Heinrich

(6) *Geology*—35 members

Buddington, A. F., *Chairman* (1954)
Allen, E. T.
Berkey, C. P.
Birch, Francis
Blackwelder, Eliot
Bowen, N. L.
Bradley, W. H.
Bucher, W. H.
Chaney, R. W.
Cloos, Ernst
Daly, R. A.
Day, A. L.
DeGolyer, E. L.
Dunbar, C. O.
Gilluly, James
Gregory, W. K.
Hess, H. H.
Hewett, D. F.
Kelley, W. P.
Knopf, Adolph
Larsen, E. S., Jr.
Leith, C. K.
Longwell, C. R.
Lovering, T. S.
Mead, W. J.
Mendenhall, W. C.
Nolan, T. B.
Palache, Charles
Reeside, J. B., Jr.
Rubey, W. W.
Ruedemann, Rudolf
Simpson, G. G.
Williams, Howel
Woodring, W. P.
Wright, F. E.

Foreign Associates

Bailey, Sir Edward
Escola, Pentti

(7) *Botany*—40 members

Jones, D. F., *Chairman* (1953)
Babcock, E. B.
Bailey, I. W.
Bailey, L. H.
Beadle, G. W.
Blakeslee, A. F.
Bonner, James
Brink, R. A.
Burkholder, P. R.
Campbell, D. H.
Chandler, W. H.
Clausen, R. E.
Cleland, R. E.
Couch, J. N.
Delbrück, Max
Dodge, B. O.
Duggar, B. M.

Fred, E. B.
Goddard, D. R.
Kaufmann, B. P.
Kunkel, L. O.
McClintock, Barbara
Mangelsdorf, P. C.
Osterhout, W. J. V.
Raper, K. B.
Rhoades, M. M.
Riker, A. J.
Robbins, W. J.
Sax, Karl
Sinnott, E. W.
Smith, Gilbert M.
Stadler, L. J.
Stakman, E. C.
Stebbins, G. L., Jr.
Thimann, K. V.
Thom, Charles
Van Niel, C. B.
Waksman, S. A.
Walker, J. C.
Went, F. W.

Foreign Associate

Kluyver, A. J.

(8) *Zoology and Anatomy*—45 members

Willier, B. H., *Chairman* (1955)
Allee, W. C.
Bartelmez, G. W.
Bigelow, H. B.
Castle, W. E.
Cleveland, L. R.
Conklin, E. G.
Corner, G. W.
Danforth, C. H.
Demerec, Milislav
Detwiler, S. R.
Dobzhansky, Theodosius
Dunn, L. C.
Goldschmidt, R. B.
Harrison, R. G.
Hartman, C. G.
Harvey, E. N.
Herrick, C. J.
Hisaw, F. L.
Hubbs, C. L.
Hutchinson, G. E.
Irwin, M. R.
Jacobs, M. H.
Kellogg, Remington
Lewis, W. H.
Metz, C. W.
Moore, C. R.
Muller, H. J.
Nicholas, J. S.
Painter, T. S.
Patterson, J. T.
Riddle, Oscar
Romer, A. S.
Schmitt, F. O.
Schrader, Franz
Smith, Philip E.
Sonneborn, T. M.
Stern, Curt
Sturtevant, A. H.
Taliaferro, W. H.
Twitty, V. C.
Weiss, Paul
Wetmore, Alexander
Wislocki, G. B.
Wright, Sewall

Foreign Associates

Fisher, R. A.
von Frisch, Karl
Hill, J. P.
Levi, Giuseppe
Watson, D. M. S.

(9) *Physiology and Biochemistry*—57 members

Hastings, A. B., *Chairman* (1954)
Anderson, R. J.
Ball, E. G.
Bard, Philip
Bronk, D. W.
Carlson, A. J.
Clark, W. M.
Clarke, H. T.
Cohn, E. J.
Cori, Carl F.
Cori, Gerty T.
Davis, Hallowell
Doisy, E. A.
DuBois, E. F.
du Vigneaud, Vincent
Elvehjem, C. A.
Erlanger, Joseph
Evans, H. M.
Fenn, W. O.
Forbes, Alexander
Fruton, J. S.
Gasser, H. S.
Hart, E. B.
Hartline, H. K.
Kendall, E. C.
King, C. G.

Lewis, H. B.
Link, K. P.
Lipmann, Fritz
Loeb, R F.
Long, C. N. H.
Lorente de Nó, R.
McCollum, E. V.
Mann, F. C.
Marshall, E. K., Jr.
Maynard, L. A.
Meek, W. J.
Northrop, J. H.
Oncley, J. L.
Peters, J. P.
Richards, A. N.
Rose, W. C.
Schmidt, C. F.
Shaffer, P. A.
Sherman, H. C.
Smith, Homer W.
Stadie, W. C.
Stanley, W. M.
Sumner, J. B.
Tatum, E. L.
Van Slyke, D. D.
Vickery, H. B.
Wald, George
Werkman, C. H.
Wiggers, C. J.
Wintersteiner, Oskar
Woolley, D. W.

Foreign Associates

Adrian, E. D.
Best, C. H.
Bordet, Jules
Dale, Sir Henry
Hill, A. V.
Houssay, B. A.
Lim, Robert K. S.
Tiselius, Arne W. K.
Winge, Öjvind

(10) *Pathology and Bacteriology*—39 members

Long, E. R., *Chairman* (1954)
Albright, Fuller
Armstrong, Charles
Avery, O. T.
Blalock, Alfred
Cannon, P. R.
Castle, W. B.
Coggeshall, L. T.
Cole, Rufus
Dochez, A. R.
Dragstedt, L. R.
Dubos, R. J.
Francis, Thomas, Jr.
Gamble, J. L.
Goodpasture, E. W.
Graham, E. A.
Heidelberger, Michael
Horsfall, F. L., Jr.
Huggins, C. B.
Little, C. C.
Loeb, Leo
Longcope, W. T.
Maxcy, K. F.
McMaster, P. D.
Meyer, K. F.
Mueller, J. Howard
Novy, F. G.
Opie, E. L.
Paul, J. R.
Rivers, T. M.
Robertson, O. H.
Rous, Peyton
Sabin, Albert B.
Sabin, Florence R.
Shope, R. E.
Tillett, W. S.
Tyzzer, E. E.
Whipple, G. H.
Wolbach, S. B.

(11) *Anthropology*—11 members

Kidder, A. V., *Chairman* (1954)
Hooton, E. A.
Kluckhohn, C. K. M.
Kroeber, A. L.
Linton, Ralph
Lothrop, S. K.
Lowie, R. H.
Schultz, A. H.
Shapiro, H. L.
Spier, Leslie
Tozzer, A. M.

Foreign Associates

Caso, Alfonso
Keith, Sir Arthur

(12) *Psychology*—23 members

Carmichael, Leonard, *Chairman* (1953)	Hunter, W. S.	Stevens, S. S.
Beach, F. A.	Köhler, Wolfgang	Stone, C. P.
Boring, E. G.	Lashley, K. S.	Terman, L. M.
Gesell, Arnold	Lindsley, D. B.	Thurstone, L. L.
Graham, C. H.	Miles, W. R.	Tolman, E. C.
Harlow, H. F.	Pillsbury, W. B.	Wever, E. G.
Hilgard, E. R.	Richter, C. P.	Woodworth, R. S.
	Skinner, B. F.	Yerkes, R. M.

Foreign Associates

Bartlett, Sir Frederic	Pieron, Henri	Thomson, Sir Godfrey

(13) *Geophysics*—20 members

Fleming, J. A., *Chairman* (1954)	Ewing, Maurice	Pekeris, C. L.
Adams, L. H.	Griggs, D. T.	Piggot, C. S.
Berkner, L. V.	Gunn, Ross	Reichelderfer, F. W.
Bjerknes, J.	Gutenberg, Beno	Rossby, C.-G.
Byerly, Perry	Iselin, C. O'D.	Slichter, L. B.
Byers, H. R.	Lambert, W. D.	Thompson, T. G.
	Macelwane, J. B.	Wulf, O. R.

Foreign Associates

Helland-Hansen, Björn	Sverdrup, H. U.
Jeffreys, H.	Vening Meinesz, F. A.

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Biographical Memoirs

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